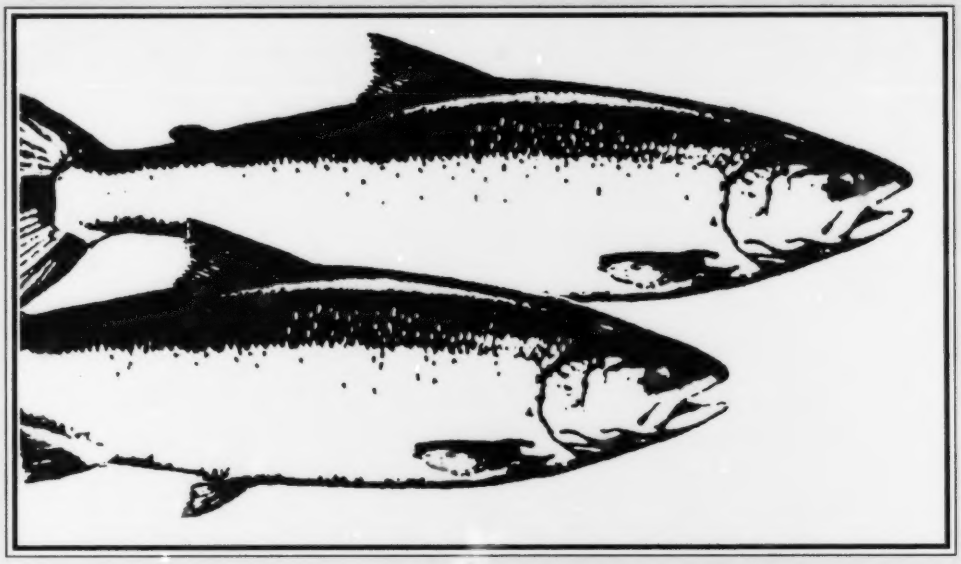
British Columbia Salmon Farming Manual



British Columbia Salmonid Disease Handbook

by Brad Hicks, D.V.M.

Published 1989 by

Province of British Columbia
Ministry of Agriculture and Fisheries
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PREFACE

This handbook was written to aid farm staff with disease identification and to increase their awareness of the variety of diseases which are present in fish.

When using this handbook, you should keep in mind that our present knowledge and understanding of fish diseases is primitive. It is important for farmers to stay abreast of developments in fish health for, as our knowledge of fish disease increases, disease diagnosis and control methods will change.

Several people and institutions have contributed to material used in this handbook. Most of the photographs were collected at the Fish Pathology Laboratory at the Ontario Veterinary College. Drs. Joan Budd, Hugh Ferguson and Joe Geraci and Messrs. Richard Moccia and Vaughan Ostland all contributed to the collection of this material. Some photo-graphs were obtained while I was employed with the B.C. Ministry of Agriculture and Fisheries.

Drs. Peter Hewitt and Al Castledine of the BCMAF were helpful and encouraging during the preparation of this handbook. Carol Lamont and Anita Pohozoff assisted with production.

Brad Hicks, D.V.M.
Vancouver
August 1989

1. BACTERIAL KIDNEY DISEASE (BKD)

INTRODUCTION

Bacterial Kidney Disease is presently the most commonly diagnosed disease affecting pen reared salmon. It is caused by a small gram positive diplobacillus, *Renibacterium salmoninarum*. Although sporadically diagnosed in all ages of fish, this disease is primarily seen in Pacific salmon after salt water entry.

GROSS PATHOLOGY

Fish with BKD may be apparently normal or they may have extensive lesions. Superficial lesions range from small sores or pimples (spawning rash) to large abscesses under the skin resulting in large soft "boils". Affected fish are often dark, with enlarged bellies and bulging eyes (exophthalmos). Hemorrhages may be seen at the base of fins and small white nodules may be present in the gills.

Lesions in the muscle range from small red spots (hemorrhages) to large, deep, blood tinged, fluid-filled abscesses. The large abscesses are most frequently seen in the epaxial muscles.

Lesions in the visceral cavity are variable. In the most dramatic cases, all of the internal organs contain variable sized, grayish white granulomas (nodules) and either a clear or cloudy fluid may be present in the visceral cavity. In less dramatic cases, single organs may be affected. Typically, whitish granulomas are seen in the heart, spleen or kidney. In the case of exophthalmos, granulomas are usually found retrobulbar (behind the eye).

A special note on spawning rash: the term spawning rash applies to the small, pimple-like lesions seen on the flank of maturing fish. These small pustules usually contain phagocytic cells engorged with *R. salmoninarum*. Diagnosis of this form of the disease is best accomplished using histopathology. This is because each lesion contains relatively few bacteria and it is often difficult to find them when examining smears using either Gram stain or IFAT. Fish with spawning rash often have none of the internal lesions usually seen with BKD. In brood rainbow trout, these superficial skin lesions tend to disappear post-spawning.

DIAGNOSIS

The gross pathology in a full blown case of BKD may be sufficient to formulate a diagnosis. Disseminated, deep-seated fungal infections are the most common differential diagnosis of these severe cases. In cases with moderate to mild gross pathology, diagnosis of BKD should be confirmed by either culture or visualization of the causative organism. (Gram stain and IFAT are the methods most commonly used). It should be noted that none of these diagnostic techniques reliably detect the bacteria below concentrations of approximately 10^4 to 10^6 bacteria per gram of tissue. Therefore a negative result on one of these tests does not indicate that bacteria are not present, it only indicates that the bacteria were not found using these relatively insensitive tests.

EPIDEMIOLOGY

BKD has been diagnosed in wild and cultured salmon and trout varying in age from young fry to broodstock. There is marked variation in the prevalence of BKD in any age group and marked differences in prevalence between hatcheries and grow out sites. We still require extensive epidemiological studies to identify the most severely affected groups. Of the species presently being cultured, the Pacific Salmon (coho, chinook) are more severely affected than Atlantic Salmon and rainbow trout. However, severe outbreaks of BKD in Atlantic salmon and rainbow trout can occur.

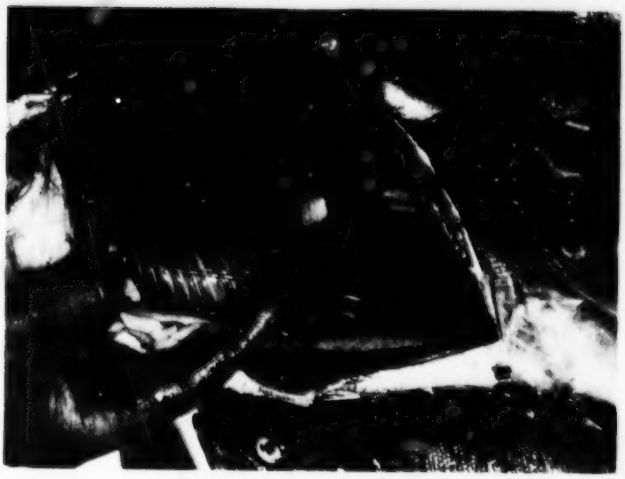
PREVENTION

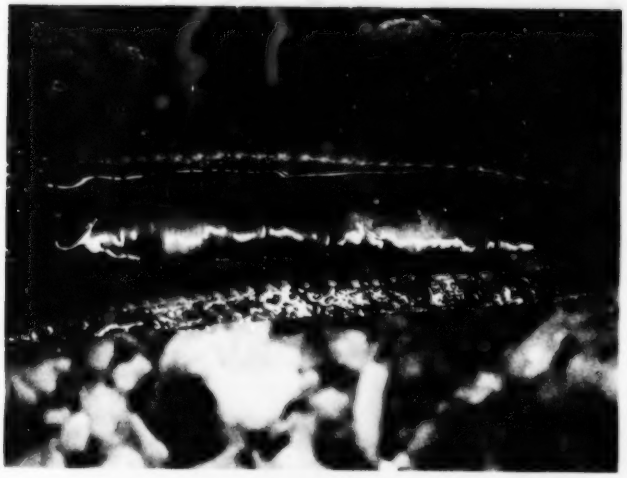
Because vertical transmission occurs with BKD, screening of ovarian fluid and rejecting positive samples has been tried in an attempt to prevent this disease. However, diagnostic tests presently being used may not be reliable enough to develop an effective screening program. As a matter of good management, eggs from fish with cloudy ovarian fluid or ovarian fluid in which BKD has been diagnosed, should be discarded.

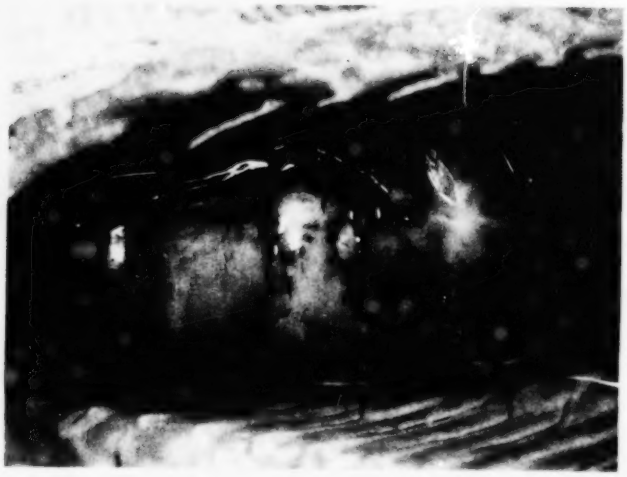
As with all other diseases, the best method for preventing BKD is the application of good husbandry techniques which provide the fish with an environment suitable for its requirements. Stress must also be kept at a minimum through the fish's entire life cycle.

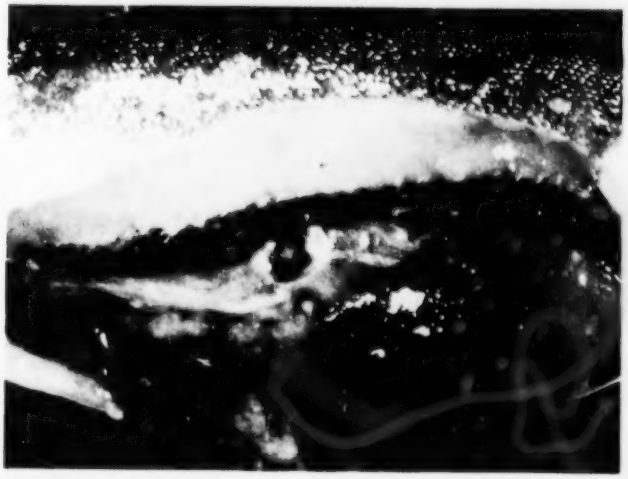
It is also prudent to examine smolts prior to transport to sea cages. A high prevalence of kidney disease in smolts may decrease their chances of performing well in sea water.

Although yet to be proven, there have been several reports of successful treatment of BKD when treatment was initiated early in the course of an outbreak. Oxytetracycline or erythromycin administered either orally or by injection have been useful in decreasing mortality due to BKD, provided treatment is initiated early.









The influence of stocking density on the prevalence of BKD is yet to be defined. However, there are early indications that decreased density has helped to alleviate the severity of BKD on some farms.

Presently several attempts at vaccinating fish for BKD are being undertaken. Once a suitable vaccine has been developed, we should see less BKD.

FIGURES

Figure 1.1 *(Upper left, page 2)*

The well circumscribed lesion in the posterior third of the kidney had a raised surface and, when cut open, contained pus-like material. Solitary lesions, as seen here, are frequently seen in fish during the subacute to chronic stages of kidney disease.

Figure 1.2 *(Middle left, page 2)*

In this fish, the entire kidney is uniformly swollen. The kidney has a slight gray cast and several small abscesses (arrows). At this stage, the fish appear to be sick and are mildly to moderately anemic. If fish in this condition are handled, you can expect increased mortality.

Figure 1.3 *(Bottom left, page 2)*

In some fish, kidney lesions are more focal. This figure illustrates localization of a kidney disease lesion to the posterior third of the kidney.

Figure 1.4 *(Upper right, page 2)*

This fish had more extensive involvement of the kidney. In this case, the inflammation and necrosis had spread throughout most of the kidney.

Figure 1.5 *(Middle right, page 2)*

This figure illustrates the maximum amount of kidney damage which can be sustained before the fish succumbs to bacterial kidney disease. The entire kidney is swollen and contains pus-like material. Fish with these extensive lesions are often small and poor doing. There is little chance of recovery for fish affected to this degree, regardless of treatment.

Figure 1.6 *(Lower right, page 2)*

This figure illustrates systemic BKD with all internal organs affected. They are swollen and contain numerous small granulomas. This represents a common presentation of BKD in severely affected fish.

Figure 1.7 *(Upper left, page 4)*

This figure illustrates the localization of BKD in the skin. Because this form of the disease is most frequently seen near spawning time, it has been called spawning rash. Each of these small pustules contains large numbers of bacteria within phagocytes. Because of the relatively small number of bacteria associated with each pustule, the diagnosis is best confirmed using histopathology.

Internally, these fish often have a normal appearance. It is difficult to find bacteria in either the kidney or heart. The relationship between spawning rash and a full blown case of BKD is yet to be established. However, we have used isolates of *Renibacterium* from spawning rash to cause typical BKD in fish held under experimental conditions.

Figure 1.8 *(Middle left, page 4)*

Another type of skin lesion associated with BKD is an ulcerative dermatitis. In this case, the affected fish usually have an advanced case of bacterial kidney disease. Histologically, the lesions associated with these ulcers are an extensive arteritis resulting in obliteration of the small arteries and subsequent infarction, necrosis and sloughing of the overlying epidermis. The ulcers themselves may be secondarily infected with myxobacteria and fungus.

Figure 1.9 *(Bottom left, page 4)*

A less frequently seen form of BKD is a miliary necrosis of the liver. In these cases, the fish usually has an advanced case of BKD in which there appears to be shedding of the bacteria into the blood stream with showers of septic emboli localizing in the liver. Histologically, these lesions range from small, septic emboli with a minimal inflammatory response to small to medium sized, well circumscribed granulomas.

Figure 1.10 *(Upper right, page 4)*

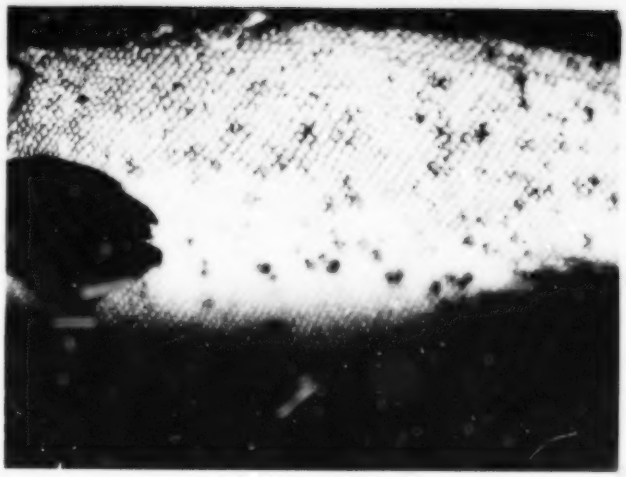
During the latter stages of BKD, we often see 1 to 2 mm focal hemorrhages throughout the skeletal muscle. Histologically, each of these hemorrhagic sites is associated with a microcolony of bacteria.

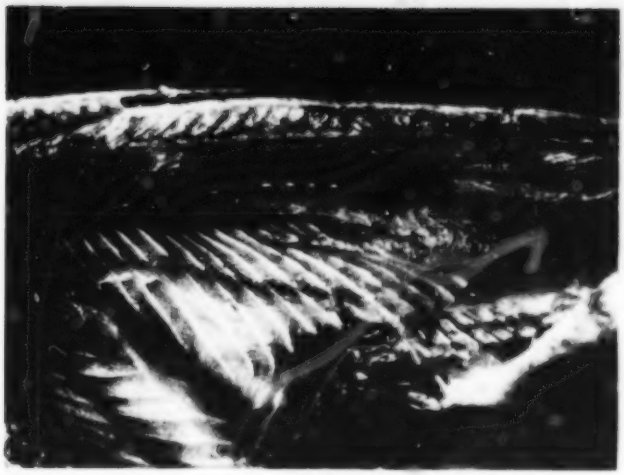
Figure 1.11 *(Middle right, page 4)*

The heart is frequently affected in chronic BKD. In this illustration, there is an extensive granulomatous epicarditis. In this case, inflammation was restricted to the outer regions of the compact layer. The only other gross pathological indication of BKD in this fish was a mildly swollen liver which was encased in a thin coat of fibrin.

Figure 1.12 *(Lower right, page 4)*

Another form of heart muscle involvement is severe granulomatous myocarditis. In this illustration there are three large, deep-seated granulomas in the myocardium. This fish also had extensive visceral lesions involving all of the internal organs.





2. FURUNCULOSIS

INTRODUCTION

Furunculosis is a relatively common bacterial disease of fish, including both salmonids and non-salmonids. Although several subspecies of the bacterium have been identified, the major isolate associated with mortality in cultured fish has been *Aeromonas salmonicida*. Furunculosis has been diagnosed in both wild and farmed salmon in British Columbia. However, to date it has been only a minor problem in farmed fish.

CLINICAL SIGNS

In acute outbreaks the first sign may be increased mortality. In less acute outbreaks, the mortality may be slow in onset and affect moderately few fish with a variable morbidity rate. In these cases, fish usually go off feed (anorexia) and develop a variety of lesions before mortality occurs.

GROSS PATHOLOGY

In acute cases, the only external pathology seen may be a slight reddening at the base of the fins, while internally the kidney and spleen may be mildly swollen and softened. In less acute outbreaks, more extensive lesions develop. Typically we see necrosis occurring in the spleen and the kidney. The lesions for which this disease gets its name are the large, liquefactive necrotic areas in the muscle. Typically, "furuncles" occur in the flank muscle, and are characterized by excavations containing blood tinged fluid. The muscle surrounding these fluid-filled vesicles is often red, soft and friable. These "furuncles" are not seen in all cases. Petechial hemorrhage has been observed on the stomach wall, swim bladder, adipose tissue, gonads and heart.

DIAGNOSIS

Because it is easy to confuse furunculosis with several other diseases, especially vibriosis, one should attempt to obtain a laboratory diagnosis whenever the signs of this disease develop. Confirmatory laboratory diagnosis is relatively simple as *A. salmonicida* produces a characteristic brown pigment when grown on TSA agar. To confirm our diagnosis, we usually use a simple slide agglutination test.

EPIDEMIOLOGY

Furunculosis is common in both salmonids and non-salmonids and has been diagnosed in all age groups. Although the disease can develop over a broad range of temperatures, it occurs mostly commonly at temperatures greater than 10°C. It has been reported in both fresh water and salt water rearing phases.

PREVENTION

As furunculosis has not been a problem in B.C.'s salmon farming industry, there has been little effort to screen smolts specifically for this disease. However, in other jurisdictions, the first line of prevention is screening of smolts. Smolts which have evidence of furunculosis are usually treated with antibiotics prior to introduction to sea water. A vaccine is also available for furunculosis.

TREATMENT

Fish with furunculosis are usually treated with antibiotics. However, *A. salmonicida* frequently develops antibiotic resistance to oxytetracycline and less frequently to potentiated sulfonamides. Therefore, a culture and sensitivity test should be done on isolates prior to deciding which antibiotic should be used for treatment. Oxolinic acid is presently the most frequently used antimicrobial for the treatment of furunculosis.

FIGURES

Figure 2.1 *(Upper left)*

Typical cases of furunculosis are seen as boils on the flanks of fish. This photograph shows a brook trout with two raised, red areas just below the lateral line. Each of these red areas represents a boil typical of furunculosis.

Figure 2.2 *(Middle left)*

On cut section, there was a large wedge shaped, red area of inflamed muscle with a zone of liquefactive necrosis below the skin (arrow). **A note of caution** - although this lesion is typical of furunculosis it does not occur in all cases of furunculosis. Both BKD and vibriosis can cause similar lesions. Therefore, it is recommended that laboratory diagnosis be obtained whenever this type of lesion is found.

Figure 2.3 *(Bottom left)*

This figure also illustrates a well developed "furuncle". The affected area is red, elevated, swollen and soft to the touch.

Figure 2.4 *(Upper right)*

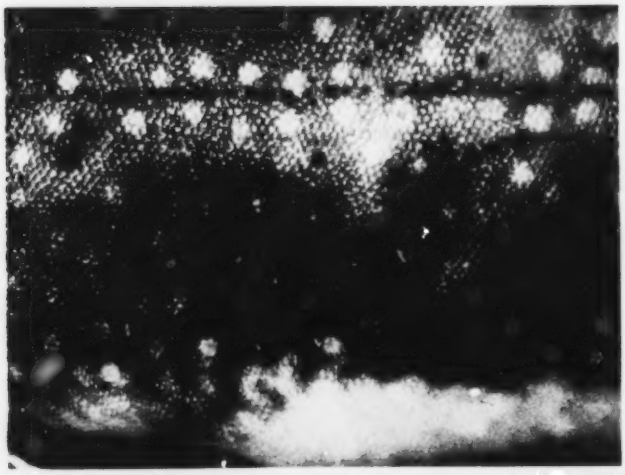
A lesion less commonly seen with furunculosis is a panophthalmitis with liquefactive necrosis (melting eye). Since this lesion is also frequently seen with vibriosis, a laboratory diagnosis is required to identify which bacteria is involved.

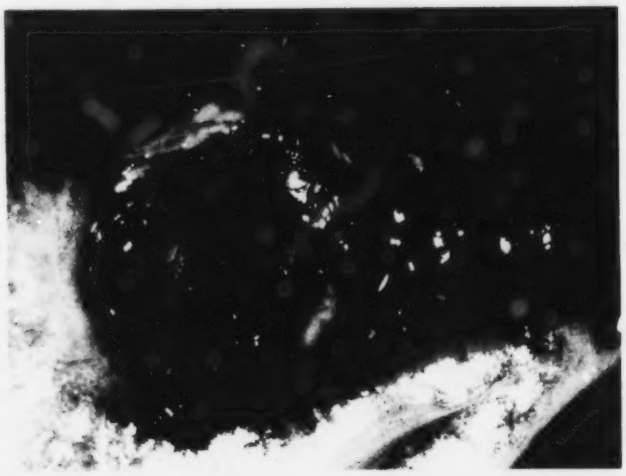
Figure 2.5 *(Middle right)*

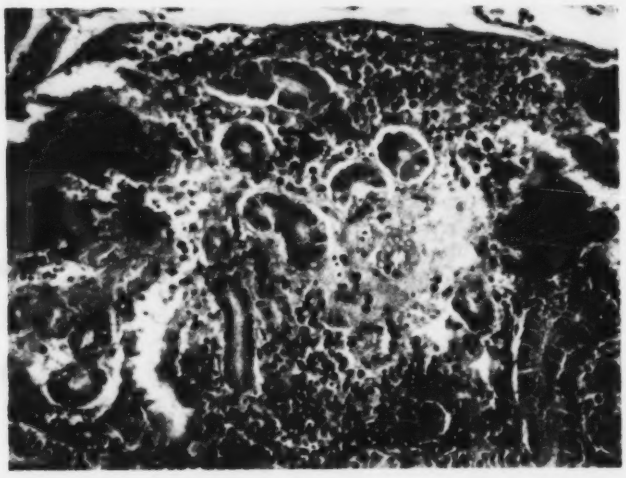
In very acute cases of furunculosis, muscle lesions with overlying skin involvement do not occur. In these cases, bacteriological and histological examination of affected fish is required. This figure illustrates the histological appearance of furunculosis in the kidney of a salmonid fingerling during an acute outbreak. There is "micro abscessation" with extensive necrosis. This type of lesion is typical of acute furunculosis and is often not noticed at necropsy.

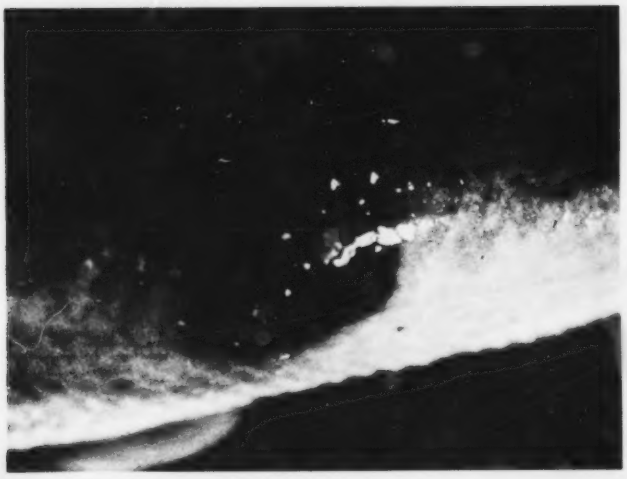
Figure 2.6 *(Bottom right)*

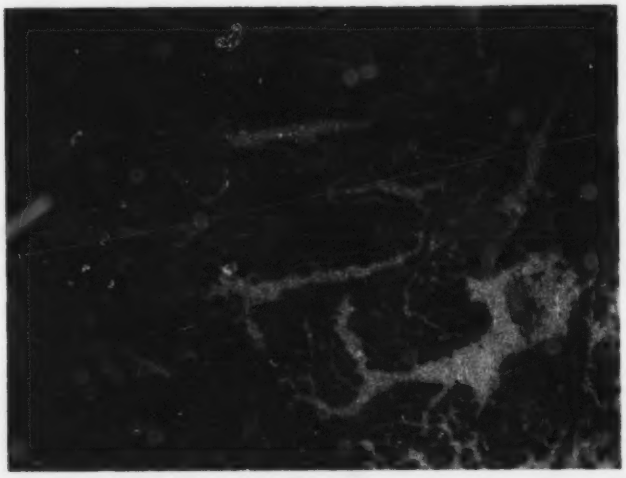
Because furunculosis is a systemic disease, there is involvement of most organs. This figure illustrates development of a bacterial colony in the spongy layer of the heart. The heart is an excellent organ to sample when attempting bacteriological confirmation of this disease.











3. VIBRIOSIS

INTRODUCTION

Vibriosis accounts for between 5% to 10% of the mortalities seen in pen-reared salmon. Vibriosis is caused by *Vibrio anguillarum* and *V. ordalli*. To date, the overwhelming number of isolates have been *V. anguillarum*.

GROSS PATHOLOGY

The gross pathology of vibriosis is very similar to that of furunculosis and hence the nickname "saltwater furunculosis". Typically, hemorrhage is seen in the skin, spleen, kidneys, liver, heart and intestine. In more acute cases, a swollen liquified spleen may be the only gross pathology. Because lesions of vibriosis are similar to those of furunculosis, a laboratory diagnosis should be obtained.

EPIDEMIOLOGY

Vibriosis has been diagnosed in most species of salmonids and in many other species of fish. Because Vibrio spp. are abundant in salt water, vibriosis is a disease which occurs during the salt water phase. It may also occur in the hatchery phase if fish are supplied with salt water. To date, the majority of outbreaks have occurred in chinook salmon. On several farms there have been no reports of vibriosis in coho. Although most frequently seen during the summer months, vibrio has occurred throughout the year. Vibriosis is most severe during the first sea year with mortalities up to 10% to 15%. In the second sea year losses are less dramatic, accounting for less than 2% of mortalities.

PREVENTION

Vaccination is perhaps the best method for preventing vibriosis. We presently have vibrio vaccines which are reported to be very efficacious. However, as with all vaccines, vaccination must be done properly for best results. Follow the manufacturer's recommendations.

TREATMENT

As with furunculosis, appropriate antibiotic treatment should be successful. Because antibiotic resistance can develop, a culture and sensitivity test should be done before treatment is initiated. Some resistance to sulfa drugs has been identified locally. The vast majority of outbreaks respond well to potentiated sulfonamides and oxytetracycline. As with other bacterial diseases, early treatment gives the best results.

FIGURES

Figure 3.1 *(Upper left)*

This figure illustrates a young smolt with an acute case of vibriosis. The red area on the flank and the large red hemorrhagic area just behind the gill cover are both signs typical of acute Vibriosis. Because furunculosis and other bacterial infections can result in similar lesions, a laboratory diagnosis should be sought to confirm your diagnosis.

Figure 3.2 *(Middle left)*

The extensive hemorrhage in the liver and a slight reddening of the rest of the viscera are common findings with vibriosis.

Figure 3.3 *(Bottom left)*

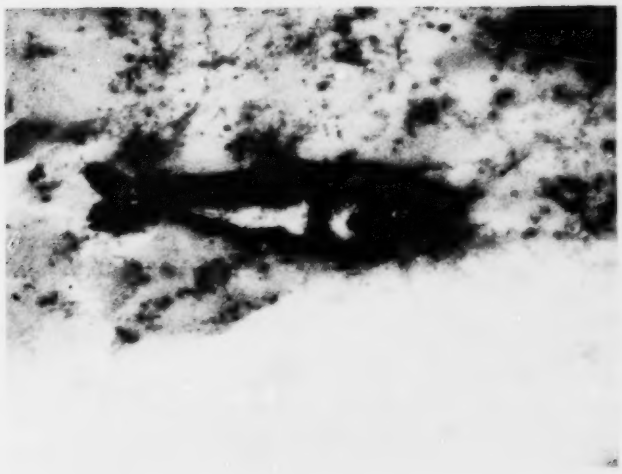
A less common, although frequently observed, lesion of vibriosis is infection of the eye. Typically, the eye becomes quite red and hemorrhagic. Eventually, the eye appears to disintegrate and often becomes infected with fungus. If we extract these eyes and examine them internally we find large numbers of vibrio bacteria. Fish with lesions at this stage do not recover from the infection.

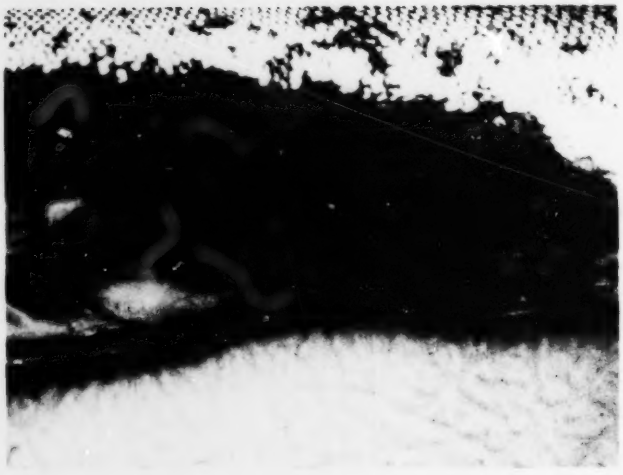
Figure 3.4 *(Upper right)*

This picture illustrates extensive purpura (small red spots) along the intestine. Vibrio and other gram negative bacteria have been associated with this lesion. Fish with this condition appear to respond well to antibiotic therapy.

Figure 3.5 *(Middle right)*

This figure illustrates an acute case of vibriosis. In this instance, we do not see any large bloody (hemorrhagic) lesions. However, there is hyperemia and congestion of the viscera. In this fish the spleen was also quite enlarged and contained red watery fluid. Liquefactive necrosis of the spleen is often the only lesion seen in peracute cases of vibriosis.





4. ENTERIC REDMOUTH

INTRODUCTION

Enteric redmouth is a disease of salmonids and non-salmonids which is characterized by the development of a reddened or hyperemic area around the jaw, hence the name redmouth. *Yersinia ruckeri* is the bacterium associated with this disease.

CLINICAL SIGNS

Peracute and acute cases of ERM have clinical signs similar to those seen with other gram negative bacterial septicemias. The fish become lethargic, go off feed and there is an increase in both morbidity and mortality. There is reddening at the base of the fins and around the vent, but most prominent around the mouth. Internally, we often see an enlarged spleen, swollen kidney, numerous hemorrhages in the lower intestine and some focal hemorrhages in the muscle. In the more chronic stages, we frequently see exophthalmos and various degrees of fin rot.

EPIDEMIOLOGY

Salmonids and non-salmonids, both wild and farmed, are susceptible. This disease is most prevalent at warmer water temperatures and in young fish which have been stressed.

PREVENTION AND TREATMENT

Good immersion vaccines are available which protect against clinical outbreaks of enteric redmouth. In enzootic areas, fish should be vaccinated. In the case of a clinical outbreak, antibiotic therapy should be initiated. In British Columbia, enteric redmouth has not been a severe problem, although some cases have been reported. Therefore, in instances where you suspect a bacterial disease, a laboratory diagnosis should be obtained.

FIGURES

Figure 4.1

The lining of the body cavity shows extensive small hemorrages and the spleen appears swollen.

Figure 4.2

There is marked reddening in an opaque cornea (eye). The mouth is also slightly reddened.

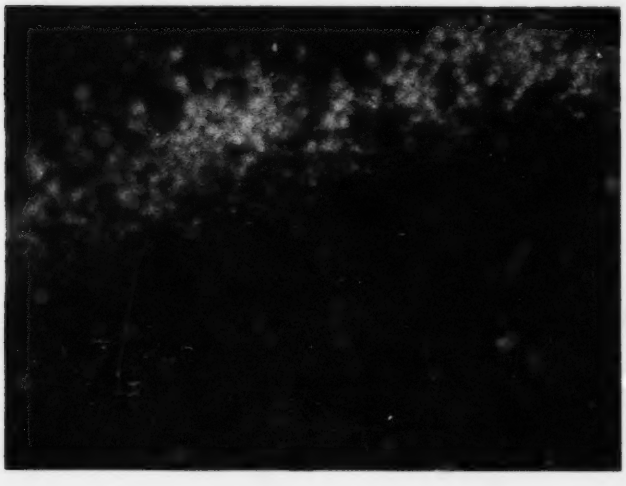


Figure 4.3

The anal fin shows obvious hemorraging, especially along the leading fin ray.

(Photos courtesy of Dr. Robert Busch, Clearsprings Trout Co.)

5. BACTERIAL GILL DISEASE (BGD)

INTRODUCTION

BGD has been diagnosed in both salmonids and non-salmonids. Historically, myxobacteria and flavobacteria in hatchery water supplies have been identified as the organisms responsible for BGD. Although the events leading to colonization of the gills by these bacteria are poorly understood; overcrowding, heavy loading with organic suspended solids, overfeeding, and high ammonia levels have all been identified as factors contributing to outbreaks of gill disease. Despite attempts to prevent gill disease by minimizing these factors, BGD does affect most hatcheries from time to time.

CLINICAL SIGNS

Fish with BGD have an increased breathing rate, go off feed, are lethargic, often swim near the surface and crowd around incoming water. There is a rapid increase in mortality.

GROSS PATHOLOGY

Gill covers are flared and the gills are puffy. In acute cases, the gills are bright red due to hyperemia. In chronic cases, gills are pale due to epithelial hyperplasia. Chronic gill disease also leads to severe weight loss.

DIAGNOSIS

Diagnosis of bacterial gill disease is based on history, clinical signs, gross appearance and microscopic findings. With whole mounts of gills, the filamentous bacteria can often be seen in very large numbers. If we smear the gill on a slide and stain it with Gram stain, we see long gram negative filamentous bacteria. Histologically, a diagnosis can be made by observation of large clumps, or stacks, of filamentous bacteria associated with the hyperplastic gill epithelium.

EPIDEMIOLOGY

Most texts describe BGD as a disease of young fish. Although most cases of gill disease are diagnosed in young fish of less than 10 grams, BGD does occur in larger fish and has been diagnosed in market-size trout. Outbreaks of BGD have been reported at temperatures ranging from 1° - 20°C.

TREATMENT

Treatment of BGD requires two steps. The first step is to remove the initiating problem; overcrowding, high organic loading in the water, etc. The second step is to treat to rid the gills of the bacteria. The most efficacious treatment to date appears to be bath treatment with Chloramine "T". This drug is added to the water at the rate of six to eight grams per 1,000 litres (6-8 ppm) for a one hour bath. Although one treatment is sufficient in uncomplicated cases, an additional one or two treatments at daily intervals may be needed in severe cases.

FIGURES

Figure 5.1 *(Upper left)*
This photograph illustrates a form of gill disease in which we see large areas of necrosis in the gill, with both bacterial and fungal involvement. This is typical of fish which have been transported without having been taken off feed. During transport, fish may regurgitate and large amounts of partly digested gut content flood the gills. The material is very damaging to the gills, causing large areas of necrosis with secondary colonization by myxobacteria and fungus.

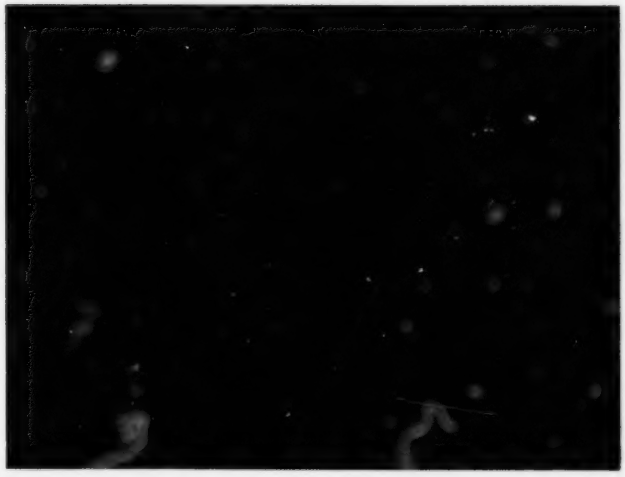
Figure 5.2 *(Middle left)*
In a more common form of BGD, the gills are pale with thickened filaments as seen in this pansize trout. Fish with the type of gill disease illustrated here respond well to treatment with Chloramine-"T".

Figure 5.3 *(Bottom left)*
This is a histologic section of the gill of a fish suffering from BGD. Note the haystack-like accumulations of bacteria coating the surface of the gills.

Figure 5.4 *(Upper right)*
This fry was suffering from typical BGD. Note the puffy gills which have forced open the gill covers.

Figure 5.5 *(Middle right)*
This is a gram stained smear (oil immersion) of the bacteria associated with BGD. In severe cases of bacterial gill disease, we can usually make a diagnosis by examining this type of smear. However, in mild and moderate cases of BGD, smears often give false negative results. In instances where we suspect BGD and yet the smear is negative, the gills should be examined histologically.

Figure 5.6 *(Bottom right)*
This figure illustrates a wet mount prepared from a fish with typical BGD. Note the long filamentous bacteria forming a large mat. It is important when examining whole mounts of gill smears from squash preparations to look very carefully for these mats of bacteria. If the gills are also heavily colonized with *Trichodina* or *Ichtyobodo* (Costia), it is possible to miss the bacteria. This results in *Ichtyobodo* or *Trichodina* being incorrectly blamed for the gill disease. If you are unsure of which agent is most significant in a particular case, histological examination will help to identify the most significant agent.



6. MOUTHROT

INTRODUCTION

Mouthrot is seen in smolts usually within six to eight weeks of introduction into the ocean. Affected smolts are usually, but not always, smaller than average. Myxobacteria *(Cytosporophaga)* can readily be isolated from dying fish.

CLINICAL SIGNS

Affected fish go off feed. The fish usually lose weight following salt water introduction and may become quite thin. There is extensive erosion and ulceration of both the lower and upper jaw and mats of necrotic yellow material are seen on the jaws. In some cases, we see reddening at the base of the fins, and some hemorrhage is seen internally along the intestine.

DIAGNOSIS

Diagnosis requires microscopic examination of mouth scrapings for the presence of long filamentous gram negative bacteria. Histological examination of this material reveals a chronic ulcerative epidermitis with colonization by the long filamentous bacterial rods.

PREVENTION AND TREATMENT

Specific techniques for prevention of this problem have not been developed, however, clinical observation suggests that selection of large, robust healthy smolts will decrease the incidence of this condition. In some instances, it appears that water temperatures above 16° - 18°C have been contributing factors. Therefore, raising fish in water below these temperatures may also be preventive.

Treatment of this condition is achieved with antibiotic therapy. The antibiotic of choice has been potentiated Sulfonamides. However, isolation of the bacteria and culture and sensitivity testing should be carried out to ensure proper antibiotic selection.

FIGURES

Figure 6.1

This recently introduced smolt has gross lesions typical of mouthrot. The yellow material associated with the ulcerative mouth lesions is made up predominantly of marine myxobacteria *(Sporocytophaga sp.)*. This condition is most frequently seen in smolts which are adapting poorly to the salt water environment, and/or have otherwise been markedly stressed.

Figure 6.2

This ventral view of the upper jaw of a fish with mouthrot illustrates the extensive ulceration of the gums. Again, these lesions are associated with marine myxobacteria.

Figure 6.3

A histological section in the sagittal plane through the lower jaw of a fish with mouthrot shows a thick mat of bacteria overlying the ulceration seen with mouthrot. There is extensive erosion of the superficial tissues and a moderately severe mixed inflammatory cell infiltrate in the underlying interstitial tissues.

7. NODULAR GILL DISEASE (NGD)

INTRODUCTION

Nodular gill disease, to the best of my knowledge, has not been diagnosed in British Columbia. However, it has been diagnosed in other regions of Canada and the United States and will likely be diagnosed in B.C. in the future. This disease is associated with the presence of an amoeba-like organism (A-cell) which is thought to cause the disease. Indeed, removal of the amoeba does result in the fish's recovery.

GROSS PATHOLOGY

Marked flaring of the gill covers, frequently with protrusion of the gills past the posterior edge of the operculum, is seen. The gills themselves are often partly or entirely fused together in obvious nodules or clumps which have a glistening or gelatinous covering. In larger fish, this covering can be scraped off without causing the gills to bleed. Fish with chronic NGD are frequently thin (pinheads).

DIAGNOSIS

Diagnosis of nodular gill disease relies on clinical signs, gross pathology and histological appearance of the gills. Clinically, fish with nodular gill disease maintain a moderately good appetite. There is rapid breathing with low chronic mortality, high morbidity rate, and a poor growth.

The gross pathology of nodular gill disease may be very similar to the gross pathology of bacterial gill disease. Therefore, it is recommended that a whole mount or a histologic section of the gill be examined in order to establish a definitive diagnosis. On whole mount gill squashes we can see large numbers of A-cells on the gill surfaces. If we look at isolated A-cells we will see them move with very delicate lamallaepodiam. Histologically, the gills are characterized by extensive, interlamellar epithelial hyperplasia resulting in fusion of adjacent the primary lamallae and adjacent filaments. Characteristically, the A-cells can be seen on the surface of affected gill lamallae.

EPIDEMIOLOGY

NGD has been diagnosed in trout in eastern Canada and in the midwestern U.S. The disease is most frequently diagnosed in fry and fingerlings. However, from time to time, it has been diagnosed in market size fish and in broodstock. This disease has been identified in fish reared in well and stream water over a wide temperature range.

PREVENTION

We presently do not know of any specific preventative measures for this condition. However, if this disease is identified in a particular group of fish, the disease can be treated with Formalin. Repeated treatments may be required to alleviate mortality.

FIGURES

Figure 7.1 *(Upper left)*

This is severe NGD in a fingerling rainbow trout illustrating fusion of adjacent lamellae into nodular groupings. On fresh specimens, the gill lamellae appear to be fused together with a gelatinous coating. This coating can frequently be scraped off as a thin film.

Figure 7.2 *(Middle left)*

In this whole mount of a NGD gill filament, fusion of the adjacent filaments into nodules is well illustrated.

Figure 7.3 *(Bottom left)*

At high power magnification (400 x) spherical organisms are seen on the gill surface. These organisms are characterized by the large numbers of dark refractile granules which they contain. On fresh wet mounts these amoeba-like organisms are quite active. You can see them moving along the gill surface and climbing over one another.

Figure 7.4 *(Upper right)*

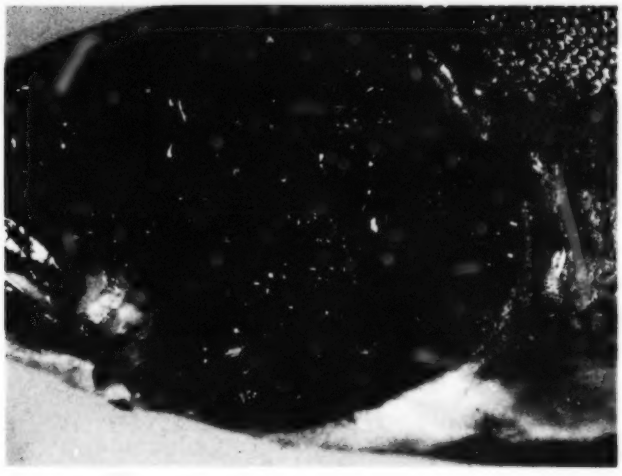
A high magnification of these organisms illustrates the distinct intracytoplasmic phagosomes. These cells have thin lamellapodia (arrow) which actively change shape in fresh whole mounts as the organism migrates. These cells are approximately 7 to 10 microns in diameter.

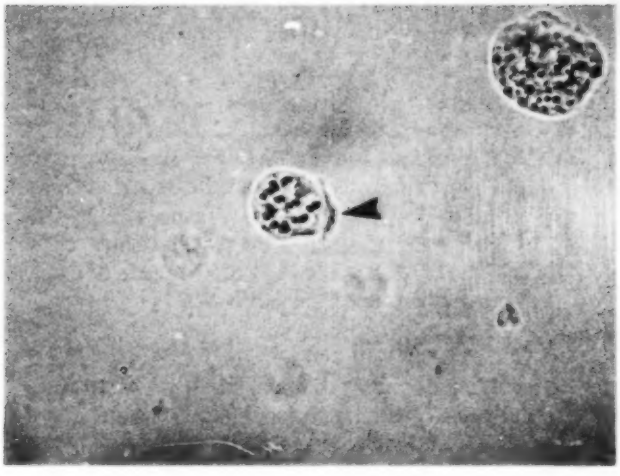
Figure 7.5 *(Middle right)*

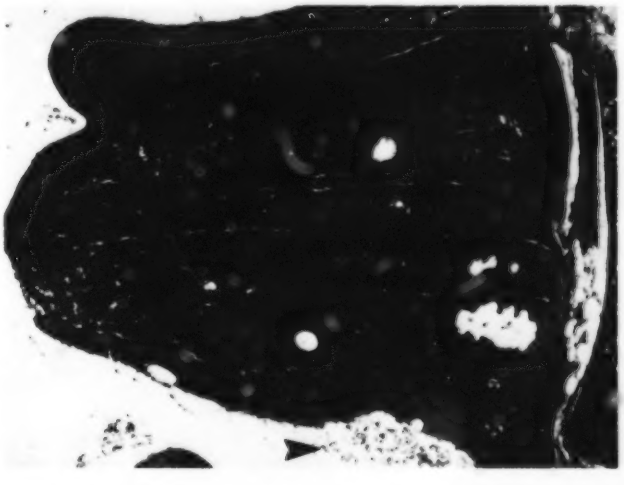
This is a low power magnification of a histologic section of a severely affected gill. At least nine gill filaments have become fused into a single nodule. The extensive epithelial hyperplasia seen in this section is typical of this disease. The amoeba-like organisms (arrow) can be seen in large numbers along the surface of the affected gill.

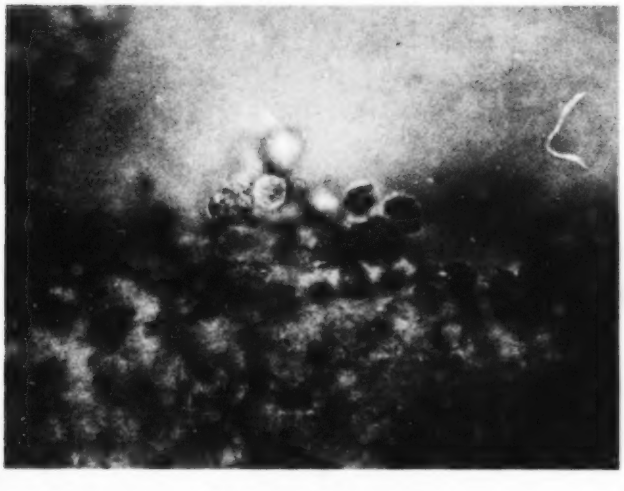
Figure 7.6 *(Bottom right)*

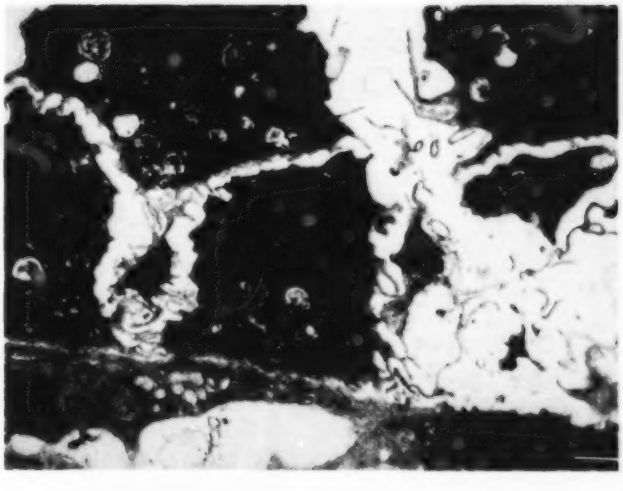
This is an electronmicrograph of the above gill. Four amoeba-like organisms are shown on the gill surface. Typically, these cells have long thin lamellapodia, many intracytoplasmic vacuoles, phagosomes and a nucleus with an outer horseshoe shaped area of dense chromosomes surrounding a less dense, paler inner core.











8. CHAETOCEROS

INTRODUCTION

Chaetoceros convolutus and other *Chaetoceros* species with spines laden with spicules are algae which, from time to time, have caused severe mortality in pen-reared salmon along the B.C. coast. Mortality is associated with relatively heavy blooms of this algae (greater than 5 cells/ml). Fish die as a result of severe gill damage which occurs when the spines of this algae become embedded in the gill tissues.

CLINICAL SIGNS

Fish suffering from exposure to *Chaetoceros* show rapid breathing and increased "coughing". Affected fish tend to ride high in the water, are lethargic and often try to escape from the affected area. This escape behavior is seen as fish swimming nose first against the side of the net pen.

GROSS PATHOLOGY

The most severely affected organ is the gill. The gills have areas of hemorrhage ranging from pinpoint to several millimeters in diameter. The gills are covered with excess mucus and have a green tinge due to the presence of the algae. Internally, the viscera are often red due to congestion.

DIAGNOSIS

Diagnosis of mortality due to *Chaetoceros* is relatively simple. The presence of large numbers of *Chaetoceros* algae in a smear from affected gills is usually sufficient evidence for a definitive diagnosis. However, if there is some doubt as to whether the *Chaetoceros* is responsible for the mortality, a histological examination will reveal the extent of the gill damage caused by the algae.

EPIDEMIOLOGY

So far, severe mortality due to *Chaetoceros* has been confirmed in most regions of the Strait of Georgia. As salmon farming expands along the coast, we may find that *Chaetoceros* will cause problems in other regions.

PREVENTION

Since we cannot prevent *Chaetoceros* blooms from occurring, we must devise ways to prevent *Chaetoceros* from causing adverse affects on our fish. Presently, the development of an early warning system to identify when *Chaetoceros* blooms are beginning has been moderately effective. Once we are aware that blooms are occurring, the fish are immediately taken off feed and left undisturbed until the threat of the bloom passes. Another obvious way to escape problems with *Chaetoceros* is to rear fish in sites where *Chaetoceros* bloom does not occur. These areas are presently being identified.

TREATMENT

Treatment for *Chaetoceros* associated problems relies on taking the fish off feed and leaving them undisturbed. If *Chaetoceros*-free water is available, it should be supplied to the fish. In the event of severe blooms, emergency slaughter may be the best solution to minimize financial loss.

FIGURES

Figure 8.1 *(Upper left)*

This figure illustrates the behavior of salmon exposed to high levels of the algae *Chaetoceros sp.* Affected fish are lethargic, ride high in the water, and have both an increased respiratory rate and a markedly increased coughing rate.

Figure 8.2 *(Middle left)*

If water with relatively few algae is supplied to the fish, they will migrate toward this source. In this case, water that is relatively free of algae is being supplied at the lower left hand corner.

Figure 8.3 *(Bottom left)*

The gills of fish exposed to high levels of *Chaetoceros sp.* are thickened, hyperemic, red and have numerous small hemorrhages. In most severe cases, they also have a greenish hue due to the presence of the algae.

Figure 8.4 *(Upper right)*

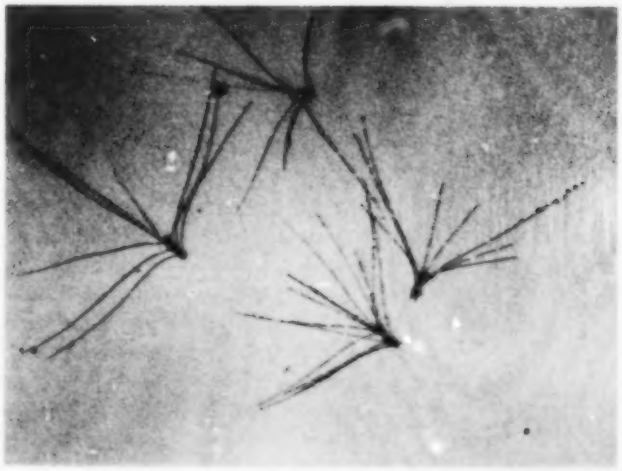
This is a low power magnification of the algae *Chaetoceros convolutus*. Depending on how the sample is handled and the plane of viewing, this algae may be seen as a single cell or a multiple celled algae.

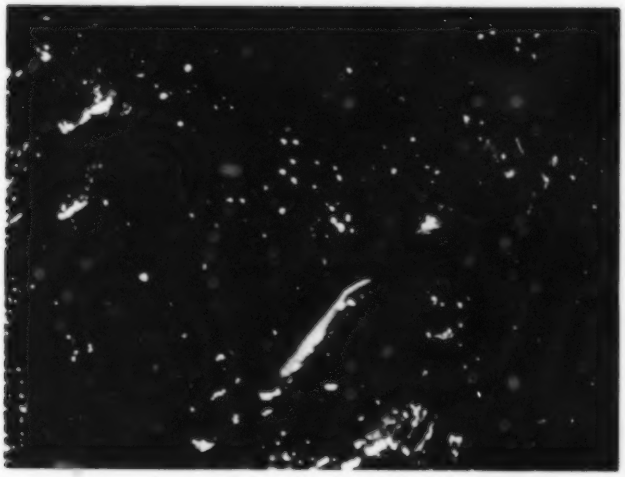
Figure 8.5 *(Middle right)*

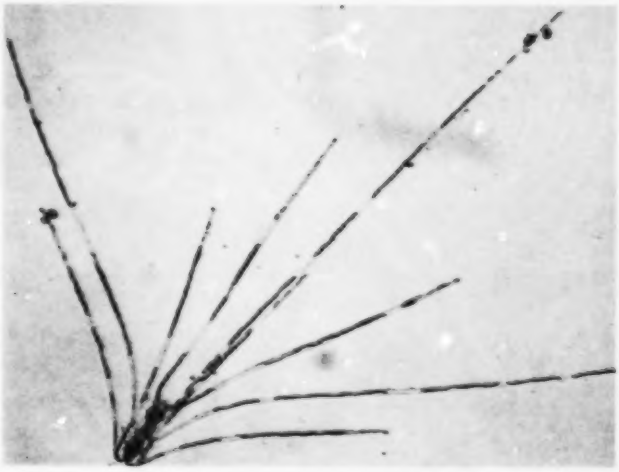
A higher magnification of *C. convolutus* illustrating the long setae which have fine barbules distributed along their surface.

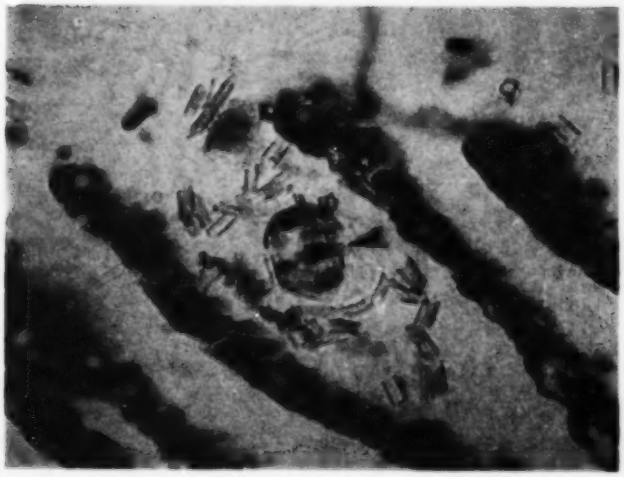
Figure 8.6 *(Bottom right)*

This histological section of a gill from a fish which died as a result of exposure to high levels of *C. convolutus* illustrates a fragmented algal cell lying between two lamellae. The large body in the centre (arrow) is the cell body of the algae. The refractile fragments surrounding this cell body are broken fragments of the setae. Careful histological examination of gills will show fragments of these setae buried deep in the interstitial tissues and frequently, setae will have worked their way deep into the cartilagenous lamella support.









9. FINROT, TAILROT, AND LIKE CONDITIONS

INTRODUCTION

Presently there is some confusion over what constitutes finrot and similar conditions. Generally speaking, these conditions are associated with long, filamentous gram negative bacteria with either flexing or gliding motion. The bacteria associated with these conditions fall under the following names: *Flexibacter, Chondrococcus, Cytophaga, Sporocytophaga,* and other bacteria generally classified as the myxobacteria. These conditions have been lumped together because they share a common overlapping pathology and prevention and treatment are similar regardless of the specific condition.

CLINICAL SIGNS

With this complex of conditions, we see a variety of frayed, worn fins which often have white highlights. There may also be patches of white, pale skin especially around the dorsal fin or along the peduncle. Affected areas are subject to secondary fungal infection. Mortality ranges from mild to severe, and morbidity is usually severe. The fish may or may not go off feed. However, as a general rule, their condition continues to deteriorate if left untreated.

GROSS PATHOLOGY

The fins, tail, back of the fish, and the mouth are the most frequently affected areas. Affected areas are dull, pale and have irregular margins. As the disease advances, these lesions often develop a yellow tinge due to the presence of massive numbers of the bacteria. Eventually, there is ulceration and frequently secondary fungal infection. In the mouth form, we often see an ulcerative gingivitis (inflammation of the gums).

EPIDEMIOLOGY

All species of salmon and many non-salmon species have been affected by this disease. Variations of this disease are seen in fresh and salt water. Fish of all ages are affected, however, we most frequently see this condition in younger fish held in crowded conditions. This disease is also more severe at temperatures greater than 10°C.

PREVENTION

The incidence of finrot and like conditions is reduced by preventing overcrowding, rough handling, poor nutrition, low oxygen and supersaturation. This disease has also been quite common in smolts recently transferred to salt water. Although the pathogenesis of this post-transfer condition is not well understood, it is thought to be most severe in smolts which have not adapted well to their new salt water environment. Again, we should strive to place good quality smolts into our seapens.

TREATMENT

Treatment of this disease has a two-pronged approach. A variety of therapeutic agents are either added to the water, and/or to the diet. From time to time, malachite green, copper sulphate, potassium permanganate, diquat, quarternary ammonia compounds and Chloramine-"T", have been used as baths. In severe cases, or instances where bath treatments are not feasible, antibiotics are added to the diet. For the most part, this disease responds well to treatments provided the predisposing circumstances mentioned above have been rectified.

FIGURES

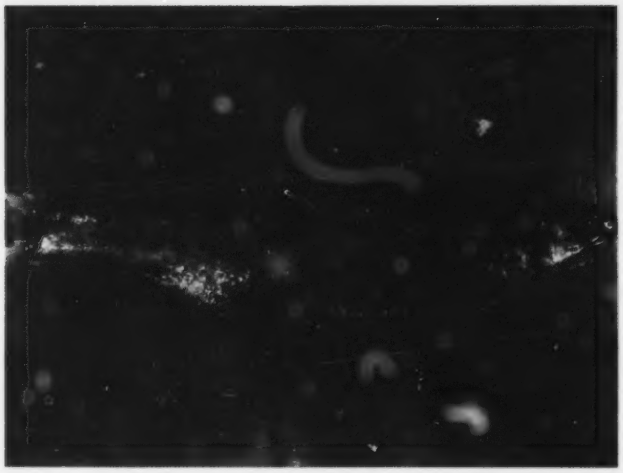


Figure 9.1

This is an example of severe tail and fin rot in a rainbow trout. Typically, long filamentous bacteria are associated with this lesion.

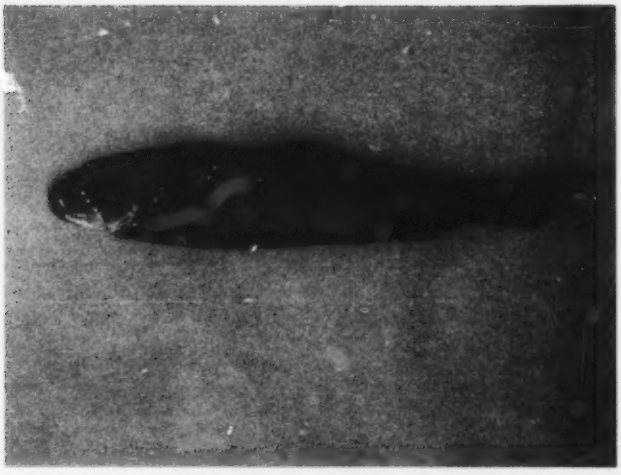


Figure 9.2

This salmonid fingerling has a classic saddle back lesion. Again, these lesions are associated with long filamentous bacteria, usually *Columnaris sp.* Histologically, there is erosion of the epidermis and the underlying dermis is heavily colonized by these filamentous bacteria.

Figure 9.3

This is an adult Pacific salmon with severe bilateral skin lesions. The bacteria associated with this lesion are also a long filamentous, gram negative bacteria. This type of lesion is seen following rough handling.

10. PROLIFERATIVE KIDNEY DISEASE (PKD)

INTRODUCTION

Proliferative kidney disease has been diagnosed in both salmonids and non-salmonids. It has been diagnosed in several locations in Europe and North America including B.C. Presently, it is not a severe problem in B.C. The causative agent is a protozoan parasite (probably a myxosporidia).

CLINICAL SIGNS

The clinical signs of PKD are quite variable. Typically, fish become dark, have a decreased appetite, and morbidity may range up to 100%. Affected fish show poor tolerance to stress and increased susceptibility to secondary infections. The mortality rate is usually low, although it is persistent and can range from 5 to 90%.

GROSS PATHOLOGY

The fish often have abdominal distention and bilateral exophthalmos. Internally, the kidney is most frequently affected. It is markedly enlarged, and contains singular and coalescing granulomas. In the most severe cases, there is also enlargement of the spleen and liver, and both of these organs may contain gray granulomas.

DIAGNOSIS

The tentative diagnosis can be based on the history of PKD on a particular farm and fish having typical gross pathology. Since the common conditions of bacterial kidney disease and nephrocalcinosis may have a very similar gross pathology, a laboratory diagnosis is advised. Confirmation depends on finding PKD cells in kidney impression smears or in histological sections of the kidney or other organs.

EPIDEMIOLOGY

PKD is generally seen in the fresh water rearing phase of salmon. The disease continues its course after the fish are placed in salt water. Although it is most frequently seen in fingerlings, it has been reported in yearling fish. Groups of fish which have had mortality as 0+, frequently had no mortality in subsequent years. This provides us with circumstantial evidence that fish develop immunity to PKD.

PREVENTION

Because PKD is generally considered to be enzootic in wild fish, it would be prudent to raise fish in water from sources where this disease is not a problem in the wild population. PKD does appear to have a seasonal periodicity. In some circumstances, this disease has been avoided by not placing the fish into river or stream fed grow-out systems until late summer or early fall. As we are only beginning to understand this disease, new methods of prevention may soon be available.

TREATMENT

To date, malachite green has been the only treatment with any degree of efficacy. These have been laboratory tests and will require field application before their usefulness in the commercial aquaculture sector is proven.

FIGURES

Figure 10.1

These salmonid fingerlings illustrate the swollen kidneys typical of PKD. Usually, the kidneys are uniformly swollen with a notable cross banding effect which is particularly obvious in the lower specimen.

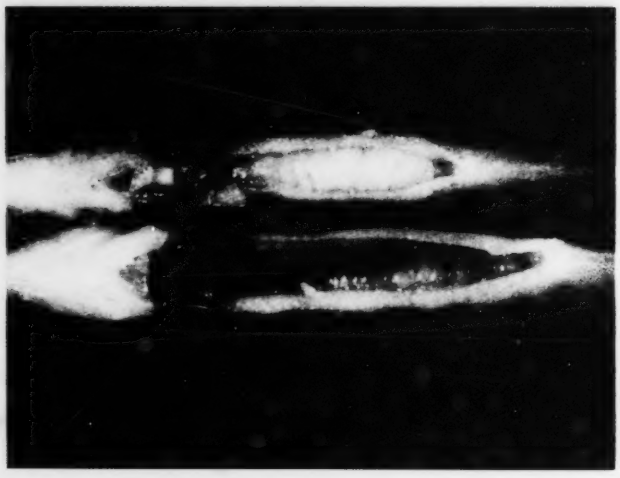


Figure 10.2

This is a histological section from the kidney of a freshwater Atlantic salmon with PKD. In the centre of the illustration, indicated by the arrow, is the protozoan parasite, associated with PKD. This disease is most reliably diagnosed using histopathology.

11. WHITESPOT ("ICH")

INTRODUCTION

Whitespot or "ICH" is caused by a ciliated protozoa, *Ichthyophthirius multifilis*. This is a very common problem in tropical fish aquariums and less common in hatchery reared salmonids. The life cycle of this parasite starts with adults leaving affected fish and dropping to the bottom of the pond or raceway. There, they undergo rapid division, resulting in 30 - 1,000 free swimming offspring. The free swimming stages then parasitize the fish and develop into adults, thus completing the cycle. This cycle takes about thirty days to be completed at 10°C and about five days at 20°C.

CLINICAL SIGNS

Fish with moderate to heavy infestations of ICH show signs of irritation ("flashing"), increased respiratory rate and decreased feeding. In the most severe cases, there will be high morbidity. Only rarely is high mortality seen.

GROSS PATHOLOGY

The pathology is pathognomonic. Gills and skin of the fish become covered with discrete, 1-2 mm. white spots.

DIAGNOSIS

Examination of skin and gill scrapings reveals a large, holotrichous protozoan with a horseshoe shaped macronucleus. Histologically, the parasite can be seen sub-epithelially.

PREVENTION

In areas where ICH is a problem, fish should be reared on fish-free water sources such as well and spring water. Also any new fish being introduced to your system should be held in quarantine with sentinel fish for 2-3 weeks to be sure that the introductions are free of ICH.

TREATMENT

Because the adult parasites live beneath the epithelium, this parasite is very difficult to treat in the adult stage. We are, therefore, restricted to treating the intermediate stages. This means that we have to treat the fish repeatedly until the last of the adult stages leave the fish. Alternate day treatments with formalin, or formalin–malachite green usually have to be repeated 3-5 times.

There is some clinical evidence that fish may become immune to this parasite. However, vaccines are not available.

FIGURES

Figure 11.1 *(Upper left)*

This picture illustrates a severe case of white spot in a fingerling salmonid. Although this disease is not common in salmonids, when severe outbreaks occur, they can result in high mortality.

Figure 11.2 *(Middle left)*

This illustrates a heavy infection in the gills. Each of the white spots in the gills is a site where the parasite is lodged beneath the epithelium. Larger fish appear to be able to sustain fairly heavy infestations. However, from time to time, such heavy infestations can kill even larger fish (200 to 800 gm.).

Figure 11.3 *(Bottom left)*

The white spots in this gill are less easily detected. This means that very careful examination of the gills is required. A small hand lens (10 X magnification) is a useful aid when examining gills.

Figure 11.4 *(Upper right)*

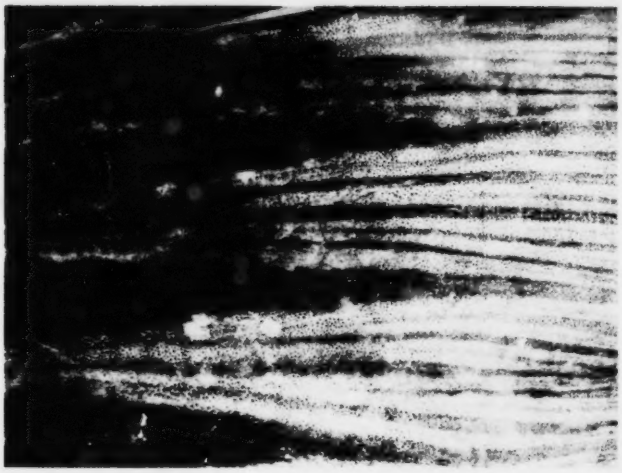
This is a close up photograph of the tail of a salmonid heavily infected with ICH. In older fish we have to examine skin and fins very carefully because these parasites are often masked by skin pigment.

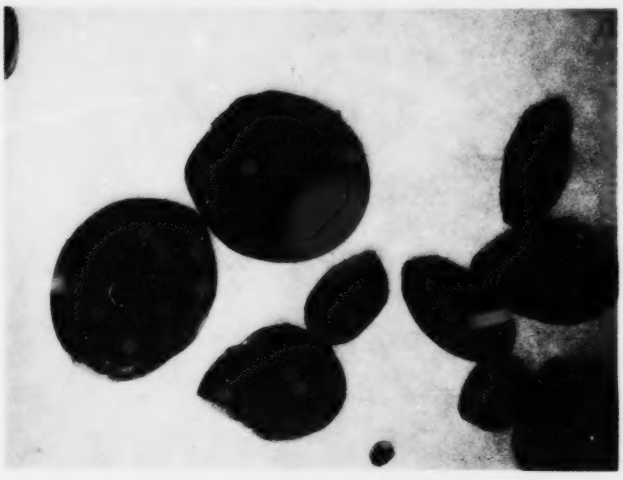
Figure 11.5 *(Middle right)*

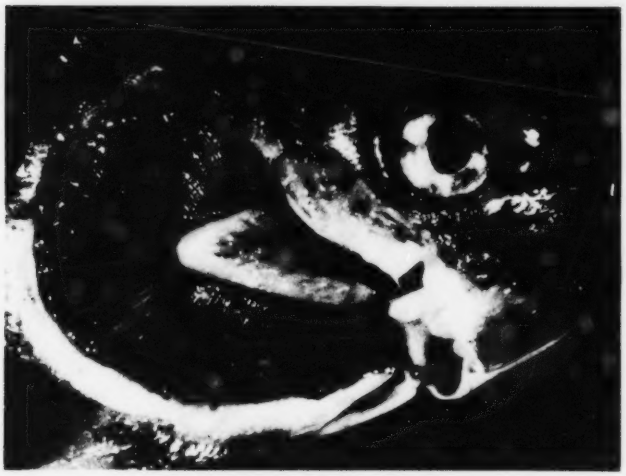
A definitive diagnosis of ICH is fairly easy to make in a skin preparation examined under low magnification. The parasite is covered in cilia (holotrichous) and contains a prominent "horseshoe-shaped" nucleus. In fresh preparations, the parasites are very active, making them easy to distinguish from other debris which is often present on heavily damaged gills.

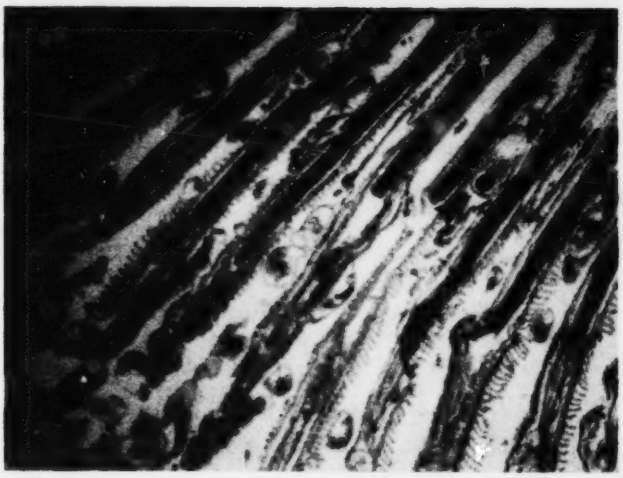
Figure 11.6 *(Bottom right)*

This is a low power photomicrograph illustrating a heavily infected gill. Note the numerous parasites in a subepithelial location along much of the gill surface. A parasite showing a typical horseshoe shaped nucleus is indicated by the arrow.









12. COPEPODS (SALMON LICE)

INTRODUCTION

Salmon lice or sea lice is the common term for copepod ectoparasites of the genus *Lepeophtheirus* and *Caliges*. Because the majority of the problems are associated with *Lepeophtheirus*, comments here will be restricted to this genus.

Infestations with a few lice is not considered to be a problem. However, infestations with greater than 25 lice and associated pathological damage, are considered to be indications for treatment.

GROSS PATHOLOGY

The gross pathology associated with lice infestation is restricted to the immediate area of infestation. In mild cases, the lice are usually present around the anal fin and the skin around their attachment is hyperemic and mildly abraded. As the number of lice increases and the condition worsens, lice are seen just posterior to the dorsal fin and mild skin ulceration begins in this area. As their numbers increase, lice migrate to the head where they cause severe erosion and ulceration. In the most severe cases, there is deep ulceration exposing the skull.

DIAGNOSIS

Diagnosis of this condition is relatively simple. The presence of large numbers of lice in ulcerated tissue around the anal fin, dorsal fin and head region is usually sufficient for a definitive diagnosis.

EPIDEMIOLOGY

Lice are commonly present on both wild and farmed stock. Generally, they are present in small numbers and do not cause any problem. To date, no severe cases have been reported in Pacific salmon and only mild to moderately severe problems have occurred in Atlantic salmon and rainbow trout.

TREATMENT

Salmon lice are treated with a one hour bath of a one part per million solution of an organophosphate. Dichlorovos is the organophosphate presently in use.

Lowering the density of salmon in pens and placing the pens where there is good flushing action also helps to lower the severity of lice infestations.

FIGURES

Figure 12.1 *(Upper left)*

This figure illustrates parasitic copepods on the gills of a salmonid. This particular copepod is called *Salmonicola sp.* These parasites are usually only a problem when large numbers are present. In the case illustrated, this is considered to be an incidental finding.

Figure 12.2*(Bottom left)*

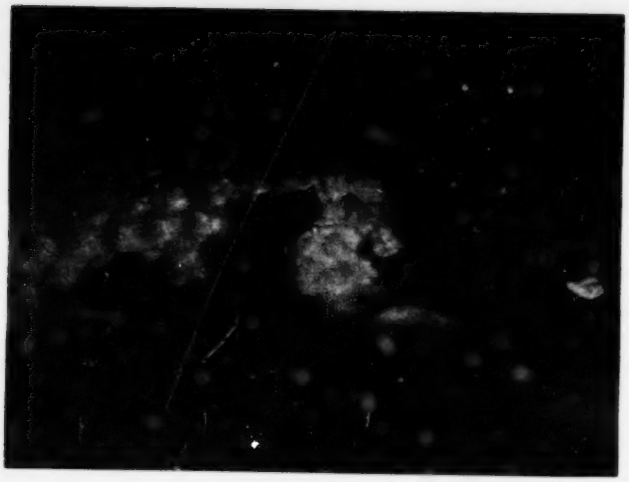
The copepod salmon louse *Lepeophtheirus salmonis* is commonly seen in Pacific and Atlantic salmon. Both wild and farmed fish carry this parasite.

Figure 12.3 *(Upper right)*

The copepod which is embedded near the dorsal fin is called anchor worm *(Lernaea sp.)*. If only a few parasites are present, there is little problem. However, if numerous parasites are present, they can be responsible for mortality.

Figure 12.4 *(Bottom right)*

This figure illustrates the clinical appearance of fish with a heavy infestation of anchor worm. The fish were milling around an incoming stream and "coughing" frequently (fish indicated by arrow).



13. TRICHODINA

INTRODUCTION

The Trichodinids are ciliated protozoa which live on the external surface of fish, especially the gills. They are commonly seen on skin scrapings and whole mount gill preparations, but they are rarely the primary cause of problems. However, when large numbers of these parasites are present on the skin and gill surfaces, they can cause considerable irritation.

CLINICAL SIGNS

In severe infestations, the irritated fish will "flash" and an increased "coughing" rate will be observed. White patches on the skin and frayed fins may develop in the most severely affected cases.

DIAGNOSIS

The condition is readily diagnosed by examination of whole mount skin scrapings or gill smears. However, because there is frequently underlying pathology associated with heavy infestations of *Trichodina* it is prudent to examine fish for other conditions whenever *Trichodina* is diagnosed.

TREATMENT

Uncomplicated infestations with Trichodina are readily treated with formalin, diquat or malachite green.

FIGURES

Figure 13.1 *(Left)*

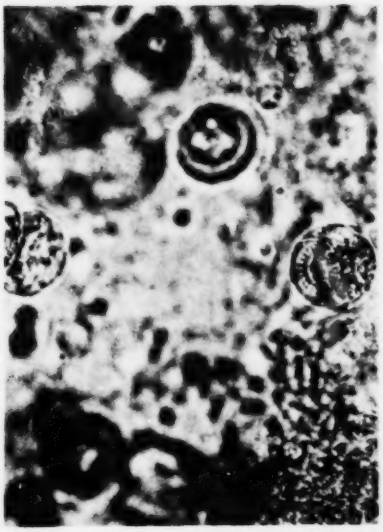
This figure illustrates numerous protozoa parasites, *Trichodina sp.*, from a wet mount of a gill. In fresh mounts, these organisms are very active, spinning on their central axis.

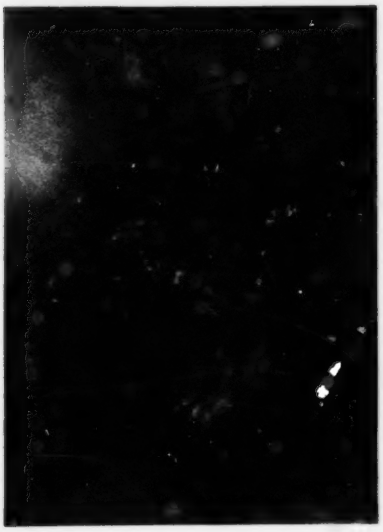
Figure 13.2 *(Middle)*

At higher magnification, *Trichodina* are recognized by the shape of their "teeth" (arrow) and the fact that they are covered with cilia. This illustration also shows long filamentous bacteria (arrowhead) typical of the bacteria associated with bacterial gill disease. In this case, the primary problem was indeed bacterial gill disease with the *Trichodina* being a secondary or associated problem. When examining whole mounts, it is very important to evaluate all findings - not just the most obvious.

Figure 13.3 *(Right)*

This is a histologic section of the nares which are heavily colonized by *Trichodina*. In section, *Trichodina* can be recognized by their size, shape, and the presence of cilia and teeth.





14. TREMATODES

14.1 Gyrodactylus

INTRODUCTION

Gyrodactylus, a monogean trematode parasite, is common in many areas of B.C. From time to time, severe infestations can occur and are most often problematic in fingerlings.

CLINICAL SIGNS

Fish with moderate to heavy infestations of *Gyrodactylus* have a moth-eaten appearance (ragged fins). They show signs of marked irritation with extensive flashing. In cases where the parasite has migrated onto the gills, there is also a marked increase in coughing. Morbidity is usually very high with severe infestations and mortality can range from minor to quite severe.

GROSS PATHOLOGY

The gross pathology associated with this disease is usually minor. There is some loss of the epithelium and there is some destruction of the outer edges of the fins. In cases of chronic infestation there is often extensive weight loss.

DIAGNOSIS

Diagnosing *Gyrodactylus* is relatively simple. Examination of skin scrapings reveals the presence of a relatively large (up to 5 mm in length), viviparous (live-bearing) trematode parasites. The haptor has two large anchor hooks and 16 marginal hooklets. There are no eye spots. A second species of monogenetic trematode which is more frequently found on the gills is *Dactylogyrus*. It can be distinguished from *Gyrodactylus* by the presence of four eye spots, one pair of anchor hooks and no embryos. *Dactylogyrus* lays eggs.

EPIDEMIOLOGY

Most species of fish are susceptible to infestations of these parasites. Usually infestations are seen in fish reared in either mud bottom ponds or raceways. This does not mean that from time to time, we do not find heavy infestations in cement or fiberglass raceways. Infestations in cultured fish are only rarely seen in fish reared on well water or water sources free of fish. If you are moving smolts into your hatchery, it is prudent that new introductions be examined for the presence of these parasites if they are problem in the area from which the smolts originated.

TREATMENT

These parasites are treated by the addition of therapeutic agents to the water. Formalin, Masoten, and Copper sulphate have been used. As these treatments are quite toxic, they must be applied carefully.

14.2 Blackspot

INTRODUCTION

Blackspot results from the deposition of melanin pigment around parasites encysted in the skin of fish. These parasites are metacercaria of digenetic trematodes. The parasite's life cycle involves a snail as the first intermediate host, fish as the second intermediate host and fish eating birds, mammals and fish as the final host. Generally, even severe infestations of blackspot do not adversely affect the overall health of the host. The major problem with this disease is decreased marketability due to undesirable black spots on the skin.

GROSS PATHOLOGY

Affected fish have obvious black spots over their entire body surface. The spots range in size from 2-4 mm and are usually elevated, giving the fish a rough texture. The spots themselves are sub-epithelial, being located in the reticular layer of the dermis.

DIAGNOSIS

A diagnosis can be made from the obvious gross pathology and by examining squash preparations of the encysted metacercaria. Definitive diagnosis to the species level usually requires completion of the life cycle in the final host and identification of the adult trematode.

EPIDEMIOLOGY

Blackspot is usually seen wherever the correct combination of final hosts and snail intermediate hosts are available to complete the life cycle.

PREVENTION

Prevention of this disease is achieved by having a water source which is either free of the snail intermediate host, or protected from contamination by the feces

of fish-eating birds. Herons, kingfishers, loons and gulls are the birds which are most commonly implicated in outbreaks of blackspot in farmed fish.

TREATMENT

There is no treatment for blackspot. Prevention is the secret. Carcasses can be salvaged by filleting and skinning the fish since the spots are confined to the skin.

FIGURES

Figure 14.1.1 *(Upper left)*

This is a close-up whole mount photograph of a fin with an attached monogean trematode *(Gyrodactylus sp.)*. These parasites are quite large, measuring one to two millimetres in length, and are relatively easy to recognize because they are viviparous (contain live young inside).

Figure 14.1.2 *(Middle left)*

This salmonid fingerling had a very heavy infestation of *Gyrodactylus sp.* The skin and fins had a "moth-eaten" appearance. Skin scrapings are very helpful for diagnosing this disease.

Figure 14.1.3 *(Bottom left)*

This is a close up illustration of the "moth-eaten" appearance of the pectoral fins.

Figure 14.2.1*(Upper right)*

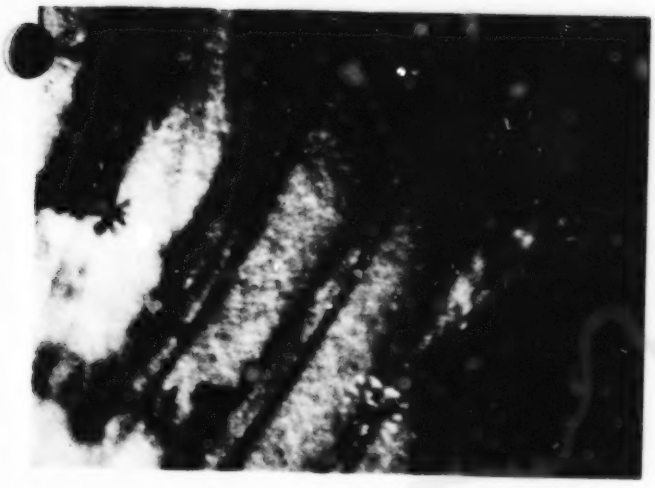
This rainbow trout had a heavy infestation of digenetic trematode parasites resulting in the condition known as "black spot". The black pigmentation is a result of melanomacrophages surrounding the encapsuled metacercaria in the dermis.

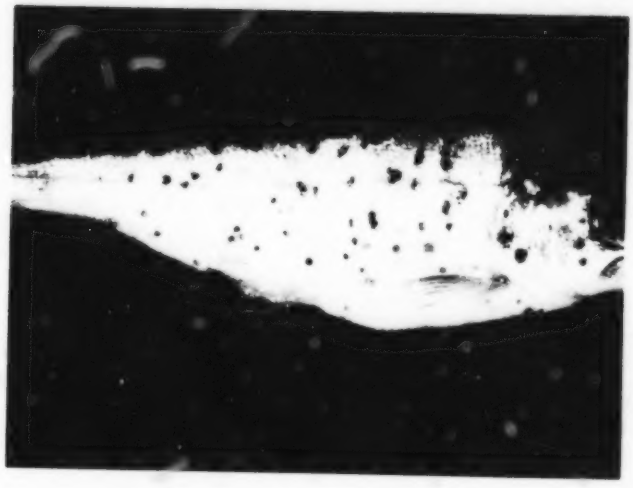
Figure 14.2.2 *(Middle right)*

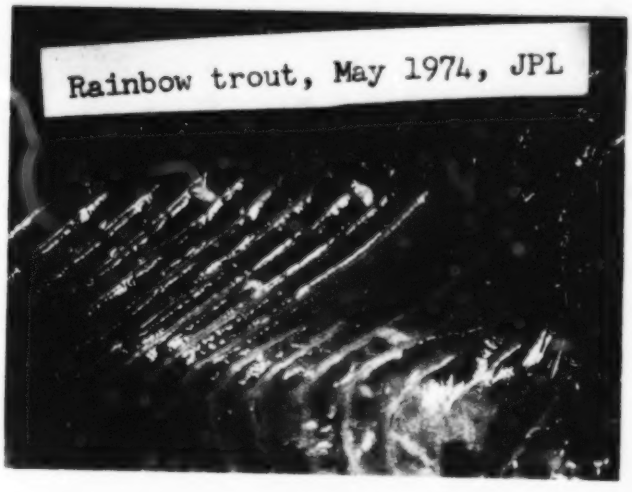
This figure illustrates the presence of digenetic metacercaria in the muscle. A condition called "yellow grub". Although these parasites are not harmful to man, they do decrease the value of the fish because of their impact on palatability.

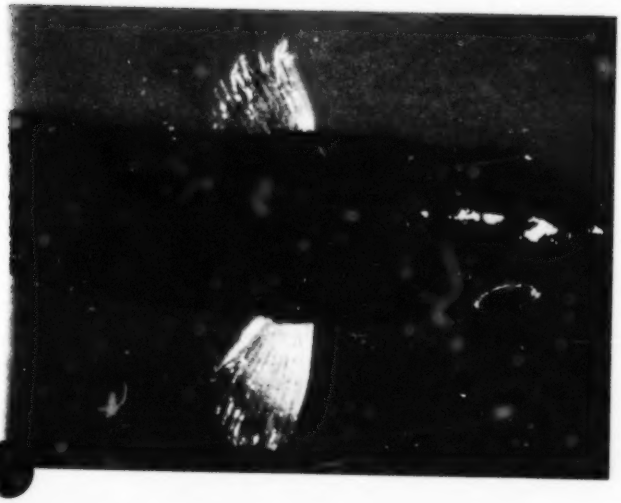
Figure 14.2.3 *(Bottom right)*

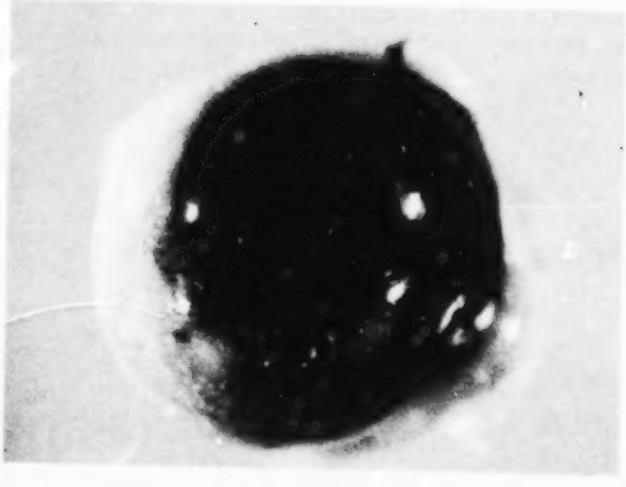
The eye from a salmonid was heavily infected with a digenetic trematode metacercaria which localizes in the cornea. Although these fish with this condition grow well, the parasites detract from marketability.






Rainbow trout, May 1974, JPL





15. NEPHROCALCINOSIS

INTRODUCTION

Nephrocalcinosis has predominantly been a problem in rainbow trout. Specifically, it has been identified in situations where there are high levels of carbon dioxide in the water or when divalent cations in the diet are not present in the correct proportions.

GROSS PATHOLOGY

Nephrocalcinosis must be distinguished from BKD and PKD. In mild cases of nephrocalcinosis, lesions are most frequently restricted to the tubular portion of the nephron. There is marked dilation of the ureters and the collecting ducts. These ducts are filled with a milky white material which has a gritty texture. In the most severe cases of nephrocalcinosis the entire kidney may become affected and it is very difficult to distinguish nephrocalcinosis from other conditions which affect the entire kidney. In these cases, histopathology is very useful in arriving at a definitive diagnosis. Histologically, there is marked dilation of the distal portions of the nephron which are packed with calcium precipitates. In instances where the nephron has ruptured, a severe foreign body reaction is seen.

PREVENTION

This condition is prevented by supplying fish with water with relatively low levels of carbon dioxide (less than 10 mg/litre) and diets containing appropriate ratios of divalent cations. Most commercially available diets do not cause problems. From, time to time, some home made diets have resulted in nephrocalcinosis. Also, as rainbow trout appear to be the most susceptible species, alternate species may be selected for propagation in areas where nephrocalcinosis is a problem.

TREATMENT

There is no known treatment for nephrocalcinosis. Prevention appears to be the only solution. Groups of fish which have severe nephrocalcinosis should be harvested as they will not perform well.

FIGURES

Figure 15.1 *(Upper left)*

This photograph illustrates a case of nephrocalcinosis in a rainbow trout. The urethra and the ureters are well delineated (arrows). In this particular example, the collecting tubules are also visible because of the calcium precipitate they contain.

Figure 15.2 *(Bottom left)*

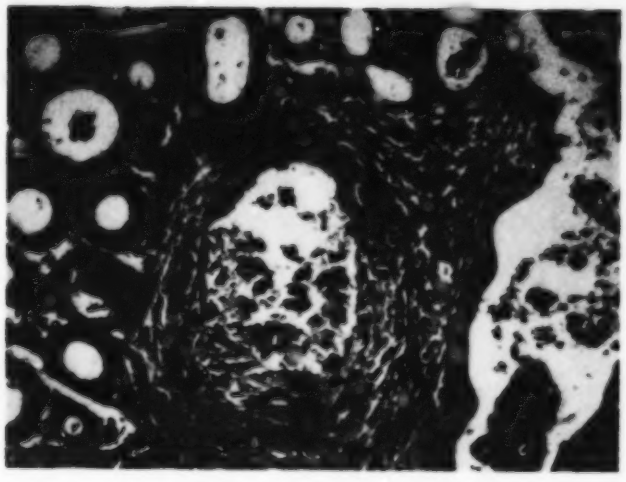
In this case of more chronic nephrocalcinosis, the ureters are markedly dilated with calcium deposits. The collecting tubules are not evident in this case.

Figure 15.3 *(Upper right)*

In this example of nephrocalcinosis, the calcium deposits are intermittent rather than continuous. One must be very careful in distinguishing nephrocalcinosis from other kidney lesions especially bacterial kidney disease and pseudokidney disease (*Lactobicillus sp.*).

Figure 15.4 *(Bottom right)*

This is a histological section of the kidney from a fish affected with nephrocalcinosis. The tubule in the centre has ruptured and a marked chronic inflammatory reaction has ensued, resulting in a granulomatous nephritis.



16. BLOAT

INTRODUCTION

Bloat, as seen in B.C.'s pen-reared Pacific salmon, results from a massive enlargement of the stomach. This condition affects all sizes of fish from recently introduced smolts to broodstock. Chinook salmon are most commonly affected. The cause of bloat is not known.

GROSS PATHOLOGY

Affected fish have a markedly enlarged abdomen and there is also muscle wasting if the condition is chronic. Internally, the stomach is markedly dilated with a very thin wall and there may be concomitant atrophy of both the pyloric caecae and the liver.

PREVENTION AND TREATMENT

This condition appears to be exacerbated by overfeeding and rapid changes in rations. When changing either pellet size or the type of diet, i.e., pelleted vs. extruded diets, the change should be gradual. In cases where bloat has developed, decreasing the feeding rate appears to alleviate most of the problem.

FIGURES

Figure 16.1 *(Upper left)*

The upper fish in this illustration shows a typical example of bloat. The belly is markedly extended, otherwise the fish has a normal appearance. The other fish in this illustration are non-smolts.

Figure 16.2 *(Bottom left)*

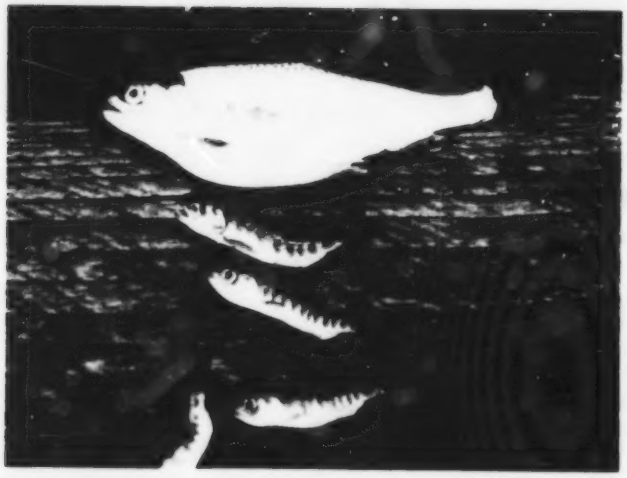
This shows the marked gastric (stomach) distention which is typical of bloat.

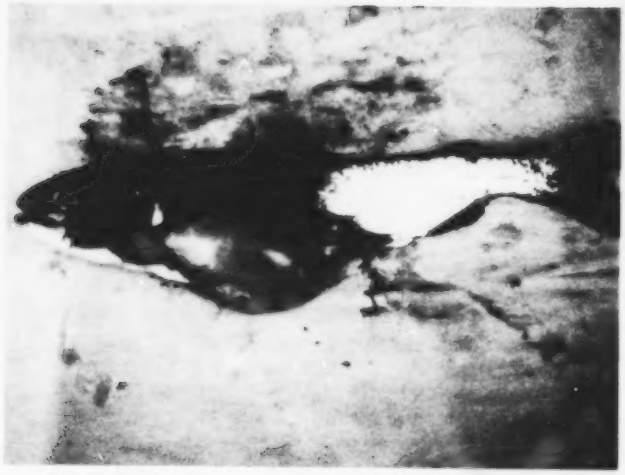
Figure 16.3 *(Upper right)*

This is another example of bloat. Note that the bloat affects the stomach to the level of the pyloric sphincter only. The pyloric caecae are not affected.

Figure 16.4 *(Bottom right)*

This is an example of bloat in a broodfish. This fish survived to maturity and was in good condition despite the presence of the markedly enlarged stomach (about 1 1/2 litres in volume). This fish died as a result of algal blooms, it did not die from the bloat.





17. MISCELLANEOUS CONDITIONS

17.1 Gas Bubble Disease (GBD)

INTRODUCTION

GBD occurs in fish being reared in water supersaturated with dissolved gasses. Although nitrogen, oxygen and carbon dioxide have all been reported to cause GBD, excess nitrogen is by far the most common cause of this condition. Conditions which promote GBD are most frequently found in hatcheries, on ground water, hatcheries with heated water and in rivers down stream from hydro-electric dams. Although all life stages of fish are affected, sac fry and "buttoned-up" fry appear to be most susceptible.

GROSS PATHOLOGY

Fish with gas bubble disease invariably develop visible pockets of gas beneath the skin and within the eyes. In sac fry, gas bubbles develop in the yolk sac. In larger fry, subcutaneous gas bubbles (emphysema) are often seen on the top of the head, along the ventral surface of the isthmus and in the roof of the mouth. In larger fish the gas bubbles are seen in the fins, under the skin in various locations, within and behind the eye (exophthalamus) and in the roof of the mouth. Internally, intravascular emboli are commonly seen in the heart and major vessels. In very severe acute cases of exposure to gas supersaturated water the fish may die before any noticeable gas bubbles develop.

DIAGNOSIS

Evidence of gas bubbles in the fish is usually sufficient to make a diagnosis of GBD. Confirmation may require measurement of the level of supersaturation in the water supply. Total gas supersaturation levels of 103% to 105% for sac fry, 105% to 115% for fry and fingerlings and 120% or more for large fish are useful rules of thumb when establishing a diagnosis of GBD.

Prolonged exposure to oxygen in excess of 250% to 300% can also cause GBD in some circumstances. When using saturation measurements to diagnose GBD there is always the possibility that by the time the measurements are made, water may no longer be saturated. In such cases, the gas bubbles may still be present in the fish.

CONDITIONS FOR DEVELOPING SUPERSATURATION

Saturation of water occurs when:

a) Excess gasses are forced into solution due to entrainment of air in the water supply.

b) Water at saturation is exposed to decreased pressure.

c) Water is heated decreasing the amount of gas which can be dissolved.

Excessive entrainment of gases in the water supply occurs when water and air are mixed together under pressure and the pressure is then released. In fish farming systems, these conditions develop when:

a) Air leaks into the intake side of pumps.

b) Air enters intakes of gravity feed lines.

c) Using venturi air injection systems.

d) Water and air mix under pressure in plunge pools.

e) Very high levels of oxygen are introduced using injection systems.

The pressure holding gases in solution is freshwater decreased when the water is drawn from wells and springs or when the water is drawn from the bottom of reservoirs and then placed in shallow tanks. This decreased pressure allows the dissolved gases to come out of solution resulting in supersaturation.

Heating hatchery water results in supersaturation because the warmer the water, the less gas it can hold. Supersaturation can result with as little as 4 degrees centigrade rise in water temperature if the water is near saturation before it is heated.

PREVENTION

Prevention requires that fish are not exposed to water with nitrogen saturation greater than 103% for young fish and 115% for older fish or oxygen saturation greater than 200 - 250%. The simplest way to ensure this occurs is to strip the water of excess gases immediately before it is delivered to the fish.

Hatchery water supplies systems should be designed and maintained to ensure that no air can leak into negative pressure lines. If water is drawn from wells or springs, it should be stripped before delivery to the fish. In situations where supersaturation can occur, the water supply should be monitored.

There are numerous devices for stripping excess gases from water supplies. The reader is referred to books on aquaculture engineering for details.

17.2 Predator Attacks

Predator attacks are a constant threat to all fish farming operations. Most fish-eating birds, mammals and fish will occasionally attack farmed fish. Predator attacks can be identified by the tell-tale trauma (bite marks) left behind by the predator. In the case of mammals, tooth marks can be seen and, in the case of birds, beak marks are frequently identified. Although the number of fish taken by predators may be minimal, many of the predators damage large numbers of fish in the process of trying to obtain a meal.

Prevention of predator attacks requires that the predator and the fish be kept separated. This is most frequently achieved by using a variety of predator guards, including nets, electric fences, guard dogs and night watchmen.

A variety of "seal-scaring" noise-making devices is available on the market. These are yet to be proven efficacious.

17.3 Lightning (Electrocution)

This is mentioned only because from time to time it is observed and it can be a baffling diagnosis. When fish are struck by lightening, the backbone is fractured at the level of the dorsal fin with concomitant rupture of the dorsal aorta in this region and the development of a large hematoma in the kidney. On farms where lightning is a problem the use of lightning arrest devices is usually helpful.

FIGURES

Figure 17.1.1 *(Upper left)*

This figure illustrates gas bubble disease in a fry. The gas bubble is obvious at the posterior end of the yolk sac. Gas bubble disease in fry of this size can occur at very low levels of supersaturation (103% and 105%).

Figure 17.1.2 *(Middle left)*

In very young salmon fry with classical gas bubble disease, the yolk sac may become engulfed in a large bubble. Fry with the severe condition illustrated here usually do not survive.

Figure 17.1.3 *(Bottom left)*

In histological sections, subepithelial emphysema is usually seen in the head region (arrow).

Figure 17.1.4 *(Upper right)*

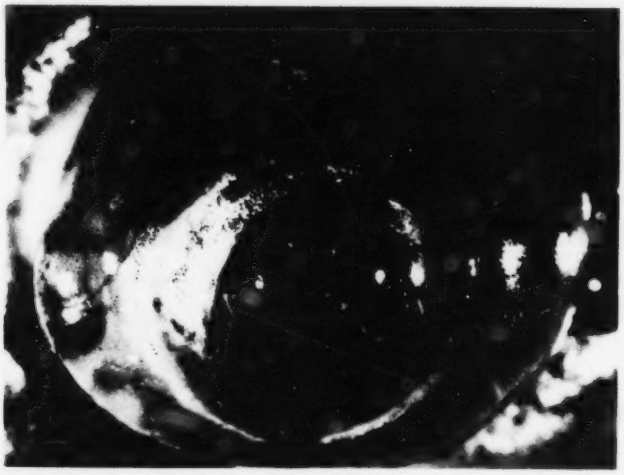
This figure illustrates intraocular (within the eye) gas bubble formation. The eyeball has become massively enlarged with several gas bubbles. Gas bubble disease of this type usually occurs at saturations of 110 to 120%. It can occur quite rapidly - this particular fish developed this problem in 24 hours.

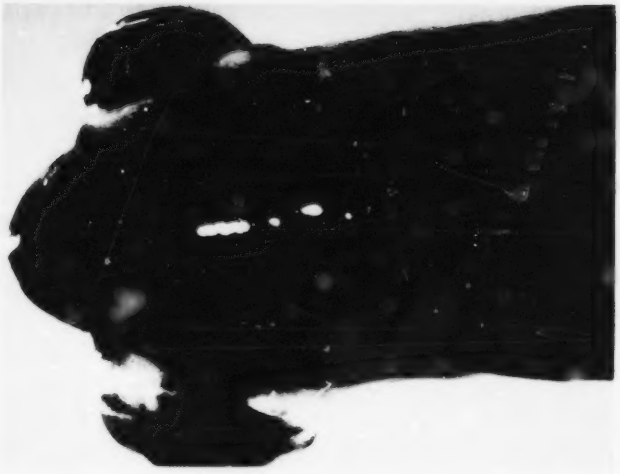
Figure 17.1.5 *(Middle right)*

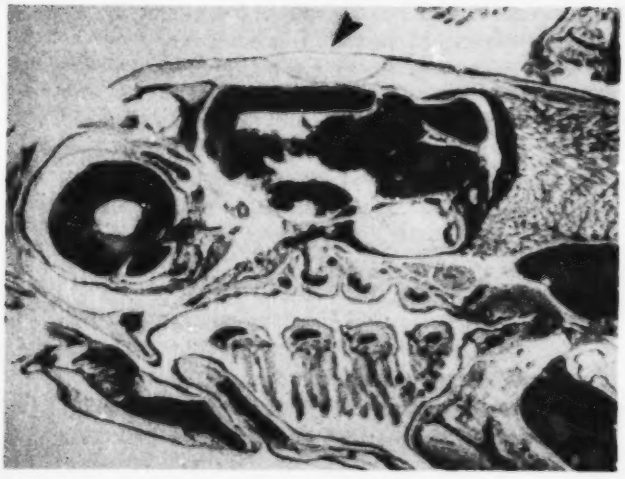
This is a dorsal view of a salmon with marked bilateral exophthalmos (bulging eyes) due to acute severe gas bubble disease.

Figure 17.1.6 *(Bottom right)*

This illustrates a frontal section through the head of the fish in Figure 5.5. Note the large intraocular, and retrobulbar, pockets of gas. Because the retrobulbar gas pockets extend into the roof of the mouth, examination of the roof of the mouth is often very helpful when examining fish for gas bubble disease.







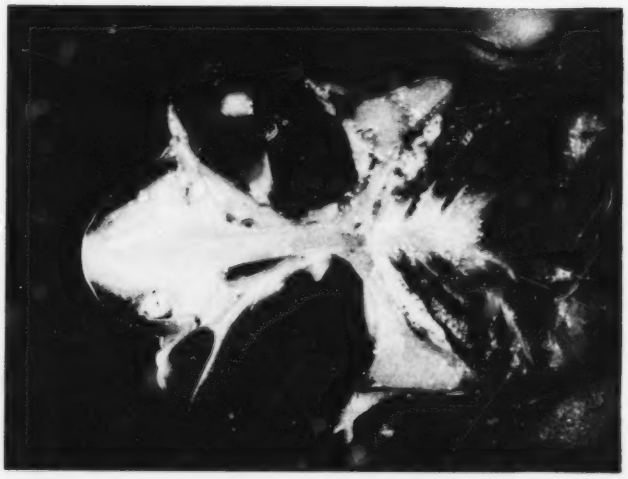


Figure 17.2.1

This is a lateral view of a fish which has succumbed to a great blue heron bite. The arrow indicates the imprint made by the heron's beak. In net pen operations, the heron will walk across a sagging guard net and fish through the netting. Because the heron cannot pull the fish back through the netting, there are repeated attacks on the same fish resulting in several beak marks along the fish's back.

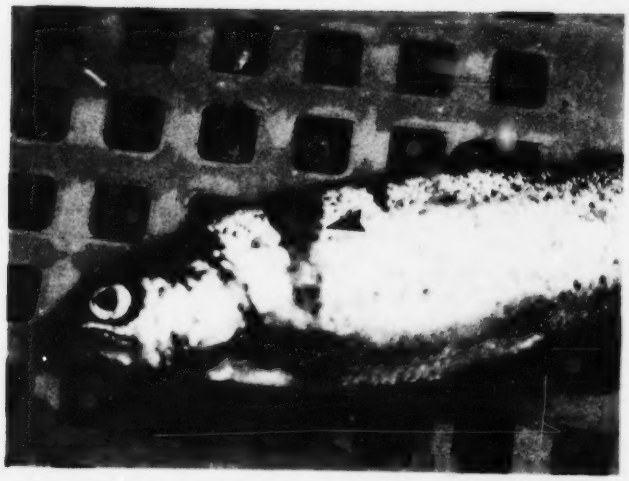
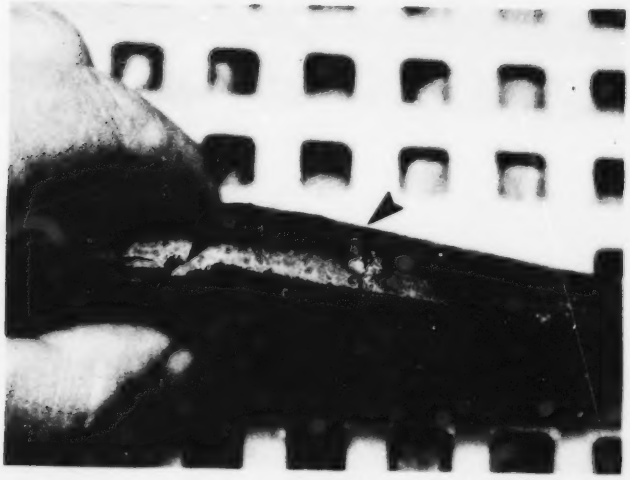


Figure 17.2.2

This is a dorsal view of the same fish showing a couple of stab wounds (arrows) from heron predation.

Figure 17.2.3

This figure illustrates slashes caused by animal bites such as seal and otter. These predators rapidly cause heavy losses by wounding many more fish than they eat.



Lightning (Electrocution)

Figure 17.3.1

This figure illustrates a fish which has been exposed to an electric shock. Typically, fish become electrocuted by lightning, various electric fishing devices, or malfunctioning electrical equipment. The obvious lesion in this case is the pale discoloration of the fish posterior to the dorsal fin.

Figure 17.3.2

This figure shows detailed dissection of the spinal column of the electrocuted fish. The backbone is fractured (arrow) and at the fracture site there has been extensive hemorrhage. The hemorrhage is obvious in the muscle and a large hematoma is present in the kidney.